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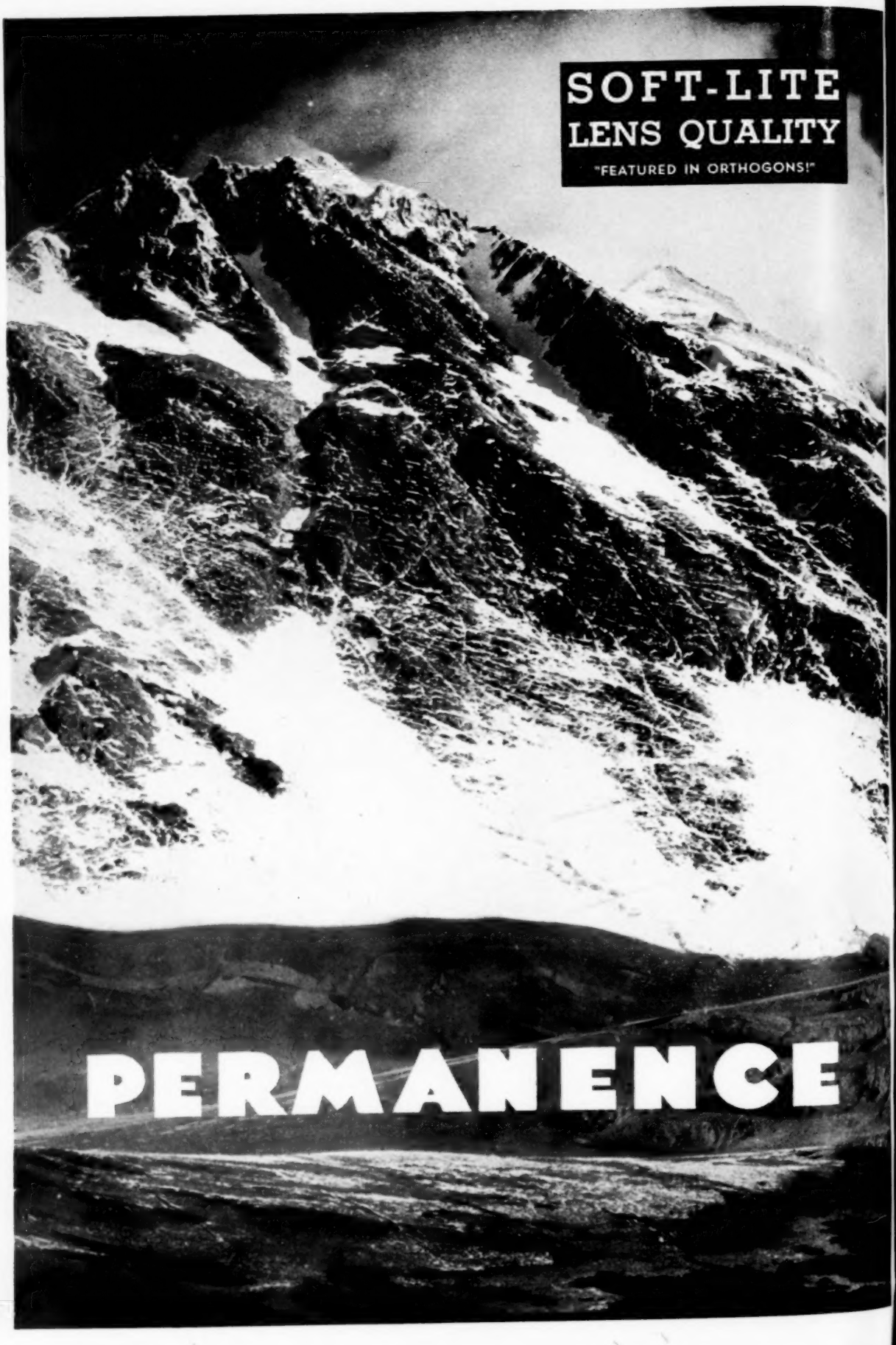
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AMERICAN JOURNAL OF OPTOMETRY

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REFRACTION AND MIGRAINE*

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Northampton,
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Introduction

The incidence of ocular migraine in the ordinary refractive practice is sufficiently high to warrant serious investigation into the question of the relation of refraction to the malady. It will be demonstrated later that four per cent of the total number of cases presenting themselves for ocular examination refractively, are sufferers from ocular migraine to a greater or lesser degree. Migraine cases examined and treated refractively in the ordinary course of practice form the subject-matter of the following paragraphs and analysis, the latter having been made with the object of endeavoring to ascertain to a reasonable degree of certainty whether refraction does or does not play an important part in providing a cure.

The collecting of the records in the cases included in the analysis has been made during a period of approximately three years. From the commencement of interest in the condition a careful questionnaire has been included in the routine used in connection with all cases examined because it soon became evident that many cases suffering from migraine were being overlooked. The description of symptoms by the patient without proper guidance is rarely worded clearly enough to enable accurate diagnosis to be made. A section of the paper will be devoted to symptoms and diagnosis.

That the refractionist is justified in considering the diagnosis and treatment of migraine as being within his province is amply demonstrated in two ways. In the first place a careful study of medical references to the complaint hardly do more than convince the reader that there is an almost total lack of agreement as to the cause or causes of migraine, and that as a consequence, no definite method of treatment has been established. Migraine is even referred to as a complaint of no serious consequence and one for which medicine has no remedy. References will be made to show that innumerable maladies and dysfunctions have been suggested as causes.

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In the second place it will be shown that a high percentage of sufferers have been completely relieved of the distressing recurrence of migraine headaches by suitable optical treatment. It will also be shown that most cases may be expected to materially benefit by wearing lenses.

The records of the cases included in the analysis have been made with the greatest possible care and with every consideration for accuracy. The collection of reports of individual cases has necessitated a good deal of work, particularly where the original prescription gave the desired relief, nevertheless out of 123 cases recognized as sufferers from migraine all but twenty-six have been seen at least twice and many three or four times. When due consideration has been given to the ninety-seven cases who have reported, it is reasonable to assume a fairly high percentage of satisfactory results from the remainder. One usually expects a more ready response to a request for a report when the treatment has failed to give the relief sought than when full relief has been obtained.

Symptoms

Quoting Gould's Medical Dictionary, Migraine is "A paroxysmal headache, usually unilateral, attended with gastric and visual disturbances." The description is somewhat too comprehensive in its terms. From observation of many cases the following would appear to be more accurate. "Paroxysmal headache, sometimes unilateral, sometimes bilateral, usually temporal and frontal, attended by symptoms of gastric disturbance and usually by visual disturbances." The visual disturbances are called "scintillating scotoma" or "fortification spectrum." Another term used is teichopsia, the meaning of which is given as, "temporary amblyopia with subjective images often an accompaniment of migraine." *(Gould)*.

Patients, upon being asked for the reason of the visit for a consultation, will, usually, somewhat vaguely refer to "terrible headaches." Rarely do they refer to the subjective symptoms in the first instances unless, again vaguely, as "mistiness." The terms will just as suitably apply, respectively, to ordinary ocular headaches and low visual acuity as they will to an attack of migraine.

After the first bad attack which is somewhat frightening, if accompanied, as it usually is, by marked disturbances in the vision, the subject appears to be more consciously concerned with the headache than with the scintillations. The latter disappear completely in the ordinary type of case in about twenty minutes. The following headache usually persists for several hours or even for the rest of the day. Sleep, often, is necessary for complete disappearance. It is somewhat natural therefore that the comparatively short period of visual disturbance should be neglected during description of symptoms especially when it is realized that the subjective phenomena is very difficult for the average patient to describe. The scotoma, except for its scintillating margin is absolute and, except for the first few seconds after the onset, is para-foveal. Experience has taught the patient that the visual disturbance always disappears,

therefore, they are subconsciously comfortable in their minds about it. They subconsciously avoid the difficulty of attempting to describe a patch of "nothingness" even when they are aware of it consciously which is not always the case. The scintillations, too, are not easy to describe. They are not always projected into the visual field as a definable object but are appreciated, mentally, as a disquieting phenomena of "flickering." The nearest analogy, would, perhaps, be in an attempt to describe the appearance of the atmosphere in an excessively hot climate, *i.e.*, the effect usually associated with the term "heat rising."

Occasionally we get migraine headaches without being able to find evidence of scintillating scotoma, but such instances are rare. In the average attack there is, first, a sensation of visual disquietude which, after one or two experiences, becomes a certain indication to the sufferer of an approaching headache. Occasionally this is preceded by a sensation of elation, a form of spurious ability to perform almost any task allotted. (The latter symptom, however, is not by any means typical of the average case). Then follows the definite central scotoma. Perhaps the word "central" is not quite correct because the whole of the fovea does not appear to be involved. "Hemianopically central" would appear to be a better term. Half of the central vision is definitely affected immediately, although it is possible, but definitely difficult to show, that the whole fovea may be involved in some cases for a few seconds. The scotoma then travels peripherally and finally disappears out of the edge of the field. The process takes, on an average, twenty minutes from beginning to end. The typical headache associated with migraine makes its appearance as the scotoma begins to disappear; it is described as a "sick headache," is often very severe and even prostrating, usually appears in the temples (one or both) develops forward (supra-orbital and frontal) and is sometimes occipital as well.

The sensation of nausea is rarely accompanied by actual vomiting in the average type of case. A percentage of cases are actually sick during an attack. There is, however, a definite sensation of gastric disturbance. Many subjects believe they are suffering from a "liverish" condition which, to them, explains all the symptoms they appreciate, *i.e.*, the visual and gastric disturbances and the headaches. The typical headache is accompanied by mental inertia. There is a desire for quietness, darkness (or dim illumination) solitude and a prone position. Sleep if obtainabile, usually brings relief. The necessity for continuation of work (physical as well as mental) during an attack predisposes to extreme and acute mental suffering. In some cases the continuation of work is visually, mentally and physically impossible.

The above descriptions of symptoms apply to the average type of case, but there are many ways in which symptoms vary in comparison between different cases. In some the headache will appear coincident with the commencement of the scotoma and scintillations. Sometimes the scintillations are very noticeable and are occasionally described as col-

ored. In other cases the scintillations will be slight with a widespread scotoma. Occasionally a case appears to suffer from a migrainous scotoma without a headache or with only a slight dull one.

Extreme cases, fortunately, are few in number compared with those of average type met with over a period. Extremity may be classed under the headings of "acute" and "chronic." Acute extremity includes cases who recover completely between attacks but are quite prostrate or almost totally incapacitated, for a longer period of time than is experienced in average cases. Visual disturbance may persist for several hours, the visual acuity may fall for extended periods to 6/60 or less and the headaches persist for several days. There may be recurring scotoma and scintillations during the course of the headache.

In the "chronic" type of extreme case the visual acuity may be reduced to 6/36 or 6/60 in one eye or both, for extended periods of days or even weeks. One case is on record in which the V.A. was reduced to 6/36 each eye for over three months. (Case 121, Plate 3). In another case nearly total blindness was experienced in one eye or the others for periods lasting up to two days. In the average type of chronic case the headache is usually continuous, although somewhat varying in intensity, and of the dull type. It is more general as regards position in the head. Any necessity for mental effort causes extreme suffering. Traffic and crowds of moving peple are avoided because if encountered, the headache devclops to an acute stage. Scintillations are not a very noticeable symptom in chronic cases, rather would one describe the condition, in so far as the subjective effect is concerned as unstable maintenance of visual perception. There is an almost total lack of possibility of visual concentration. The condition has usually developed as progressive from the average type of intermittent paroxysmal headache and scintillating scotoma with a history of increasing frequency and severity.

A migrainous scotoma may be a factor of danger to the affected person during an attack. It may also, in certain circumstances, constitute a danger to other people. If, for instance, the driver of a motor car, without realizing the significance of a migrainous scotoma in his visual perception, as is very likely to be the case, has an attack while traveling he might easily run down a pedestrian or cyclist without seeing him. The scotoma might even be extensive enough to cause the oversight of another car and result in a collision. A case of severe scintillating scotoma (see case 31, plate 1) who suffered from very frequent attacks, gave up driving his car for a long period because he learnt by the experience of several narrow escapes, that he was not always safe when driving. Another case (see case 123, plate 3), stated that during an attack he could, by rotating his head, either see or not seen an approaching car. He had learned to stop by the side of the road until the scotoma passed. Recently on the Watling Street between Fenny Stratford and Towcester a motor cyclist was reported to have crashed into the back of a stationary car, fortunately escaping with minor injuries. He declared afterwards that

he did not see the car although looking straight ahead. A migrainous scotoma would easily explain the possibility of such an occurrence.

In moderate or even mild cases the visual acuity is nearly always slightly subnormal in one eye or perhaps both, at the end of an examination even with the best ametropic correction in place. It is not so much a matter of inability to read the normal line of type as an inability to read it without hesitation or correctly throughout. It is as though the visual acuity varies intermittently and rapidly—too rapidly to permit of smooth reading. There is a lack of inclination in making the effort to read below the line short to normal. Cases overlooked as migrainous during the questionnaire have been suspected and afterwards confirmed as migrainous by this symptom. In some cases the visual acuity varies one day from another without any apparent cause.

Many patients will describe during the early stages of an attack, a condition of hemianopic blindness; when reading, only half a word is visible at a time; of two objects, close together, only one may be seen when the other is "fixed."

In different cases different conditions of particular visual activity will precipitate an attack. One case on record could always bring on an attack by looking at a naked paraffin lamp flame. There was a suspicion that the odor of paraffin was associated although the odor without the flame had a negative effect. Several cases have stated that passing a row of white railings while traveling in a moving vehicle would bring on an attack. Glare is a common precipitating associated cause. In many cases, however, no particular relationship of visual activity could be ascertained as associated with an attack.

On only one occasion during the period covered by the following analysis has it been possible to obtain a campimetric record of the scotoma associated with migraine. A reproduction of the charts is shown in Fig. 1. The case was a typical one, suffering from frequent attacks. The scotoma had a scintillating margin, the enclosed area being quite blind to the one degree white target used. The record was made on the Bausch & Lomb Stereo Campimeter. The area of the scotoma probably extended to the edge of the peripheral field although, to enable a rapid record of the progress of the scotoma to be made this was necessarily neglected. The "danger point" from a road safety point of view, is shown in the second record (five minutes after onset). Here is a relatively large homonymous area is blind. The scotoma had completely disappeared in twenty minutes.

Extracts and References

The medical history of a large majority of the cases included in the following analysis has led to the assumption that migrainous sufferers have found little or no relief from medical remedies. Many had subjected themselves to extended courses of treatment, others had sought relief in many and varied directions. Some had been bluntly told there was no

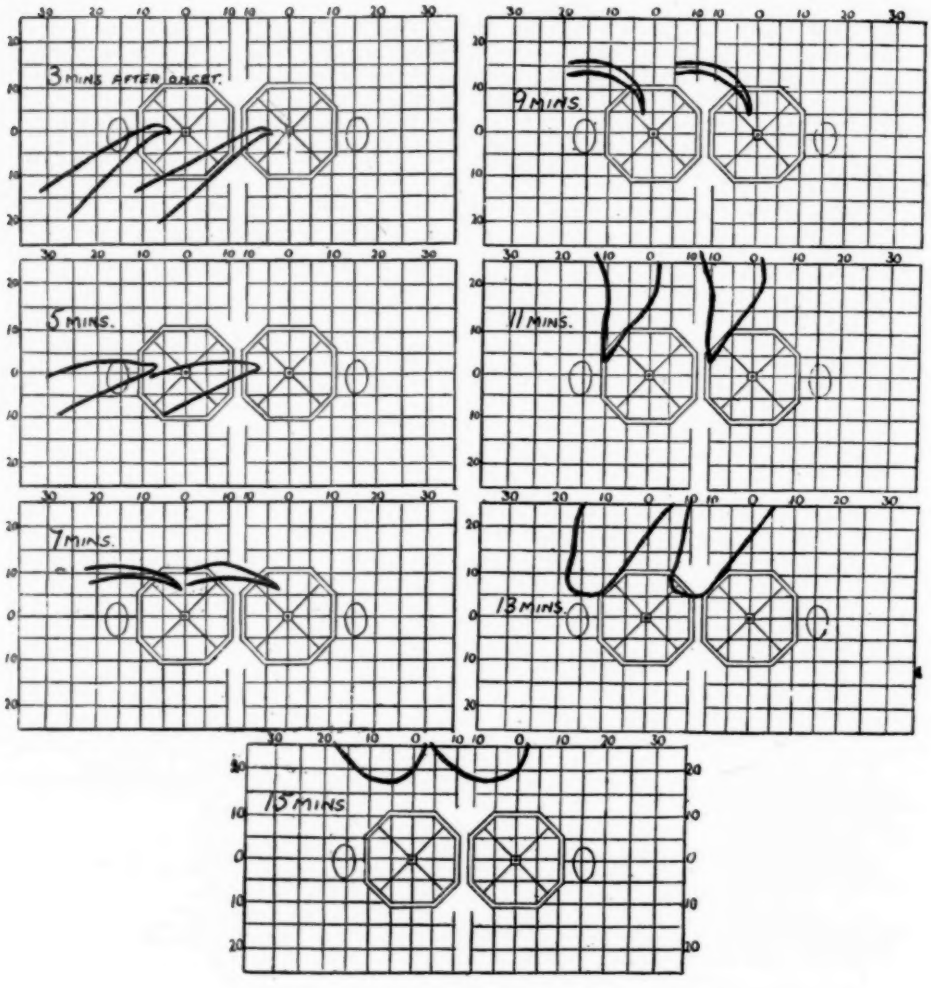

Fig 1

cure with the result that they had come to accept the recurrence of attacks with resignation. Indeed, the impression gained by almost all migrainous sufferers, as a result of their own experience and their failure to obtain relief, appears to be that their condition is incurable.

Failure to find a cure would appear to be due to the failure to ascertain the cause. Many causes of migraine have been suggested or assumed, but no evidence appears to be forthcoming that any of the suggestions have been proved to be other than associated conditions in migrainous cases. The only certain factors are the sufferings of the subjects.

The following references have been extracted from various journals and publications. They are made, more particularly, to draw attention to

the number of maladies and dysfunctions which have been suggested as causing migraine.

In a recent contribution on migraine, Drs. Critchley and Ferguson[1] make reference to a long list of associated conditions and dysfunctions, the inference being that they may be looked upon as possible causes of, or precipitating factors in migraine:

Abnormality of gastro-intestinal tract.
Liver dysfunction.
Dyskinesia of the gall bladder.
Duodenal stasis.
Any article of food.
Hunger.
Over-eating.
Inherited or acquired defect in protein metabolism.
Abnormalities in the uric acid metabolism.
Chronic nephritis.
Anaphylactic shock.
Localised intracranial oedema.
Errors of refraction and slight muscle balance defects.
Non-articular rheumatism.
Brachial neuritis.
Occiptio parietal Angioma.
Congenital cerebral aneurysm.
Congenital stenosis of the foramen of Munro.
Hyperthyroidism.
Hypothyroidism.
Ovarian dysfunction.
Pitutary secretions.
Menstruation.
Sinus disease.
Radiological abnormalities of the sella turcica.
Psychological factors—mental over-work, anxiety, suppressed rage and humiliation.
Eyestrain—front seats in a cinema, reading in poor light, microscopy.

The authors state that they consider that the importance of errors of refraction and muscle balance defects have been over-estimated.

They also assert that the essential causation remains obscure, although there are two factors "central" and "peripheral." They are inclined to regard migraine as a vasomoter manifestation associated with spasm of the cerebral arteries due to stimulation of sympathetic fibres either as a result of direct pressure, or *via* the endocrine system, occurring in a susceptible person with a migrainous constitution with precipitating factors—metabolic, alimentary ocular, sinus, etc. An editorial comment in "The Lancet" of 28/1/33, on migraine, states " . . . It must be confessed that neither Drs. Critchley and Ferguson nor Dr. Riley* offer the sufferer much hope of either prevention or cure. Neither prophylaxis nor treatment is likely to be completely successful until the causation of migraine is better understood." Also " . . . There is therefore considerable evidence that migrainous headache is associated with abnormal dilatation of the arteries of the head . . . If the immediate cause of the headache in migraine is a temporary functional disturbance of the cranial blood supply, how is this produced? The distinction of clinical types of migraine made by Critchley and Ferguson is likely to be

*Dr. Riley published a review of all aspects of the disorder with some 400 references, in the Bull. Neurol. Inst. of New York, 1932, ii. 429.

misleading if it is supposed that these types necessarily appear in different people. The same patient may at different times experience a large variety of attacks, headache, vomiting, ocular disturbances, paraesthesiae, and mild confusion occurring in all possible combinations and also as isolated symptoms; and the attack may be traceable on different occasions to eye-strain, dietary indiscretion, menstruation, exposure to cold, or emotional stress. It is clear that all causes of migraine must possess in common the power to evoke a certain unknown state of the organism which is the immediate cause of the attack."

G. W. Bray[2] says that there is evidence to prove that there is an allergic basis in many cases of migraine, the commonest causative allergens being foods. He also states that migraine is common in mothers of asthmatic children.

C. P. Symonds[3] speaking before the Royal Society of Medicine in 1927 said that the liability to migraine is hereditary with precipitating causes such as worry, excitement, diet, acidosis, etc. He states that eye-strain is rarely an important cause of attacks. He suggests a vascular spasm of the cerebral cortex particularly those parts in which vision and sensation are represented. There is (he proceeds), strictly speaking, no cure, but efficient medical treatment could diminish attacks and while nothing but good could come from correction of errors of refraction he agreed with R. Foster Moore (1925) that there was no direct casual relationship between any ocular abnormality, refractive or otherwise and migraine.

A. F. Hurst (in the discussion which followed) believed that the most common and important factor which increased the irritability of the central nervous system that produced migraine, was eyestrain. J. K. Barton suggested that troublesome head and eye symptoms were cured by spectacles but that such cases rarely exhibited signs that made up true migraine. Agnes Savill believed migraine was of toxic origin and that refractive errors were only a contributory cause.

T. C. Hunt[4] made a study of sixty cases, forty of which he describes as a bilious type, with the object of attempting to determine (1) to what extent the bilious type of migraine could be distinguished from other types, (2) how far in such cases any disorder of the liver or gall bladder can be demonstrated and whether such disorder is to be regarded as a primary cause or a secondary result of attacks of migraine, (3) to what extent treatment to correct such disorders may be expected to influence the frequency and severity of migraine attacks. He remarks upon a proportion of migrainous patients suffering from gall-stones. His conclusions were: (1) Investigation showed some evidence of gall-bladder or hepatic disorder in seventeen out of twenty-seven selected cases, (2) a higher proportion of migraine patients show gall-bladder disorder than normal subjects, (3) local gall-bladder dysfunction or disease is not a cause of migraine but may occur as the result of repeated migraine attacks.

(4) hepatic dysfunction was the cause of migraine attacks in certain cases.

(The suggestion that migraine attacks might be the cause of other dysfunctions is interesting. This is probably true of many other associated conditions frequently cited as probable causes of migraine.)

R. M. Balyeat[5] deals with the disorder chiefly as a manifestation of allergy. He favors the theory that the symptoms are due to localized cerebral oedema rather than to cerebral vascular spasm. He makes an interesting suggestion that where there may be two precipitating causes attention to one of them may result in relief from migraine attacks.

Louis Jacques[6] describes a case (according to the symptoms given) of typical prostrating migraine. He gives a full description of the refractive condition (obviously one of latent hyperopia) mentions the correction given, and states that wearing the correction cured the disorder. Curiously enough, having shown the case to be one of migraine by his description of typical symptoms he entitles his discussion of it with "A case of so-called migraine."

Crookshank[7] states that although meticulous care in general health treatment and in the wearing of glasses when they are necessary may bring relief, (and in his opinion slight degrees of hyperopia or of myopia associated with astigmatism or with imbalance, are nearly always present) in spite of this relief the migranous patient does not become *well* unless the psychological situation is elucidated and adjusted.

Investigation into the psychological state of migrainous subjects always produces evidence of mal-adjustment to circumstances. They feel subconsciously that they have failed in the management of their lives and they attempt to escape from the resultant difficulties by means of the brain-storm (which is what migraine is) which provides them with an excuse for their failure.

This brain-storm is a defense, flight and excuse mechanism in the face of difficulty determined by organ-inferiority in the shape of an affection or imperfection of the optic tract. A feeling of inability to rise to the occasion brings on an attack and then blame for failure is attributed to this organ-inferiority.

G. M. Gould[8] thinks that ninety per cent of migraine cases are immediately curable by suitable glasses and that a large proportion of the rest are also curable in time and when the secondary systemic functional effects have been overcome. It is when these secondary effects have become organic or chronic that is impossible to effect a cure.

J. H. Fisher[9] thinks that a periodic temporary swelling accompanying functional over-activity of the pituitary body explains migraine better than any other hypothesis. He believes that decompression of the sella turcica would be efficacious in the treatment of it.

S. Auerbach[10] thinks that the predisposition to migraine depends upon a disproportion between the cranial capacity and brain volume and

that attacks are brought on by exciting causes such as emotional disturbances, mental over-exertion, alcohol, sexual excesses, constipation, the menstrual period, diseases of the mucuous membrane of the nose, etc., which, through the action of the vasomotor mechanism, are calculated to exaggerate this disproportion.

W. H. Crisp[11] while emphasizing that attempts to relieve migraine should always include an examination for an uncorrected refractive error, states that in a great many cases migraine arises from extraocular sources, without apparent relation to eyestrain. He reports the treatment of twenty-five migrainous patients wih intravenous injections of peptone resulting in improvement in eighty-four per cent of the cases; ten cases were relieved by the elimination of specific foods; forty-two cases in which foods to which the patients reacted in protein sensitivity tests were wholly eliminated from the diet resulted in complete relief from migraine in fourteen cases, partial relief in nineteen, *i.e.*, some beneficial result in seventy-eight per cent of cases.

Baker[12] states that excessive use of normal eyes can be the cause of migraine and that eyestrain is the commonest cause of it. Of 100 migrainous patients for whom he prescribed glasses, 55 were cured, 31 greatly benefited and seldom had recurrent attacks, 14 were not benefited by correcting the error of refraction but five of them were cured by muscular tenotomy, one by the use of a pessary, leaving eight uncured.

C. P. Small[13] states that it is questionable whether glasses, relief of heterophoria, etc., exercises much influence over the course of migraine in more than ten per cent of the cases.

Inference from Extracts

The conclusions which may be drawn from the observations of writers on the subject of migraine are (1) that the cause or causes of migraine have not been definitely established, (2) that very many associated conditions or dysfunctions have been noted and suggested as causes, (3) that no particular method of treatment so far suggested may be expected to result in a cure or do more than give partial relief either in frequency or severity, (4) that there is reasonable agreement that an attack of migraine is caused by or associated with a secondary condition of abnormality of blood supply to the brain, particularly that part associated with the senses, (5) that there is some evidence in support of the suggestion that some of the associated dysfunctions are secondary to migraine rather than causes, (6) that errors or refraction and ocular imbalances are generally classed as precipitating factors, rather than causes, when they are present.

Refractive Errors as a Cause

The exclusion of refractive errors as an important cause of migraine must be accepted as a reasonable one based, as it is, upon such evidence as has hitherto been available, the evidence being procured from the observation of migrainous cases who have sought and experienced the aid of

lenses. That a large proportion of migraine sufferers have worn lenses cannot be denied, neither can it be said that all or many of them in the ordinary course of events, have benefited to any extent, in the reduction of the number of migraine attacks and their severity, as a result of wearing lenses. Yet it is the object of this paper to show that attention to errors of refraction may result, not only in a marked reduction of frequency and severity, but, in many cases, in a complete cure. If the analysis to be discussed below does show this then two points are proved (1) that errors of refraction are a major cause, or at least an important precipitating factor in cases of migraine, (2) that conventional methods used in providing a correction for refractive errors are inadequate and based upon an incomplete knowledge of the true refractive and associated conditions in migraine cases.

The investigation, then, becomes one which includes serious consideration of latency as well as of manifest conditions. By "latency" we must cover a broader field than is usually conveyed by the term. Actually we must include not only hyperopia, in the general and accepted sense, but also astigmatism, myopia, all forms of auto-inhibited over-activity of nerve impulses and energies and, as well, established habit factors which have been unconsciously formed by the individual in the interests of clear single vision. Simple attention to manifest ocular conditions leaves undiscovered many latent abnormal activities which are the true cause, in many cases, of migraine.

Ocular habit factors, other than normal ones, are to be found in all cases possessing errors of refraction. They are formed in a process of adaptation to the abnormal refractive condition in the interests of clear single vision and frequently, if not always, other hitherto normal conditions and activities become abnormal and are established as secondary conditions. Many secondary conditions may be more evident, in particular cases, than are the primary conditions at the moment of observation. There is always a danger of accepting a secondary abnormality as a primary condition and if the treatment of a case is based upon such an acceptance there is a probability of lack of relief, in part or whole, of the symptoms and conditions causing the individual to seek advice and help. It may even be possible that treatment based upon observation of secondary conditions without full knowledge of the primary cause of them may only succeed in aggravating the symptoms and conditions of complaint.

Secondary conditions include all departures from an accepted standard of normality of extrinsic muscular activity when those departures are as a result of conditioning reflexes from abnormal or subnormal ciliary activity. Abnormal or subnormal ciliary activity is in turn a secondary condition resulting from abnormal refraction which becomes the primary cause. The establishment of a secondary condition of extrinsic abnormality may, of necessity, in the interests of single vision, demand an inhibitive process of further conditioning which in certain cases sets

up a backward conditioning reflex in the direction of the ciliary activities resulting in a further modification of refraction. Hence a form of vicious circle, an establishment of a spurious condition of balance which becomes the nearest approach to normality of visual activity obtainable by an individual without artificial aids.

The word "balance," in the ocular sense, has unfortunately been restricted to apply more particularly to demonstrable states of extrinsic muscular activity resulting in a tendency to think of and discuss "phorias" as separate and distinct factors rather than associated conditions. To quote an instance; accommodative hyperphoria has, as a consequence been dealt with as a separate physiological state and an original or primary condition, whereas it is a secondary condition and as such should be used as a factor in diagnosing its cause which is abnormal ciliary activity. Accommodative esophorias and exophorias, due to abnormal accommodative activity are, again, prone to discussion as original conditions instead of secondary ones resulting in possible overlooking of an important, although not necessarily obvious, refractive condition. There is also the possibility of cyclophoria existing as a secondary accommodative condition. Any 'phoria, to be of value in the patient's interest must be classed as either (1) pathological, (2) physiological or (3) accommodative. The great majority are in the last group and as such are valuable data in diagnosis of true refractive conditions. The discussion of ocular balance must, to be completely successful, include refraction, refraction differences, ciliary activity, unequal binocular cilary activity, unequal monocular ciliary activity (so called sectional accommodation), extrinsic muscular activities, auto-inhibition of nerve energy, unequal sized retinal images, suppressions, suspensions and in fact any departure from an accepted standard of normality of visual activity, physiologically, optically functionally, mentally and psychologically.

Treatment of Migraine

The successful treatment of migraine in cases where errors of refraction are an important precipitating factor, the majority appearing to come into this class, is a process of establishing by artificial means, a condition of ocular normality of activity, or, to use the word in its comprehensive sense, a state of balance. It is attention to latent conditions which are, more often than not, predominatingly monocular in extent. It is, in effect, the artificial inhibition of over activity of muscles and the associated wastage of nerve energy or, perhaps a better way of putting it, the artificial inhibition of excessive nerve energy supply in place of auto-inhibition. It is the replacement of a spurious internal balance by a true balance accomplished by suitable lenses.

The exact manner in which an imbalanced condition of ocular activity produces or precipitates the scintillations and headaches in migraine is obscure. If the immediate cause of the scintillation and headaches is due to abnormal blood supply to the head, or to parts of the brain, and this appears to be supported by evidence, there is, at least, as much

argument in favor of the predisposing cause of such a condition being due to eyestrain as anything else, particularly when evidence, such as is offered in the following analysis, is available. Further, the connection between eyestrain and the sickness associated in some cases of migraine, also with numbness of the extremities, is equally obscure. It seems possible that migraine is due to two or more widely differing causes one of which is eyestrain, the latter being an associated condition in a majority of cases, and that the removal of one of the causes results in a cessation of attacks. At the same time there appears to be a danger of classing dysfunctions as causes while there is as much reason to call them results. For instance, many sufferers have definitely stated that digestive disorders have disappeared after cessation of migraine attacks following the prescription of suitable lenses.

It is reasonable to suggest that the therapeutic value of lenses is only partly understood. At the same time their real, properly understood value can only be known after extended investigation into the reactions following their use. This should form a profitable opening for research.

Interest in the possible cause of migraine being due to refractive errors was accidental. A particular case, under attention for refraction, was persistently esophoric, the esophoria being of a low amount. The patient, seen previously many times, was consistently uncomfortable, in his visual activities, in spite of every effort in the prescribing of lenses made conventionally. He was a migraine sufferer, the attacks appearing two or three times each week although, at the time, the migraine was not considered to be associated with his ocular condition. He was in the habit of taking frequent doses of aspirin, phenacetin and liver salts. The ocular condition included over stimulation of the internal recti. Other extrinsic muscle tests showed, on different occasions, varying amounts of cyclophoria and some hyperopia. It was decided to experiment with inhibitive prisms and a total of four prism dioptres, base in, was included, empirically, in his previous sphero-cylindrical correction. The distortion, due to the prisms disappeared in three days. In a few weeks the report was that not only had the ocular distress disappeared but that there has not been a single attack of migraine in the period. To find the possible association of inhibition by base-in prisms the original lenses were worn for a short period on about six subsequent occasions. Each time the old glasses were used a bad attack of migraine appeared within forty-eight hours. No attacks were experienced during the wearing of the prisms. Subsequent examinations failed to show either cyclophoria or hyperphoria. The reading distance lateral balance had become normally exophoric. Slight changes in the manifest refraction were found but more alteration was found in one eye than in the other. It is now fairly clear that the manifest condition, previous to the wearing of the prisms, was not the true refractive condition and that it represented the result of internal effort to esablish an approximation of normal balance refractively and muscularly, in spite of the errors of refraction; the result

being the maintenance of a spurious balance on a high plane of stress. The case has now been consistently free from migraine for four years.

Following this, cases possessing symptoms of migraine were, for a period, all given an amount of base-in prism in their correction and the results carefully watched. The number reporting either complete relief from migraine or marked reduction in frequency and severity of attack was remarkably encouraging. A system of classifying migraine cases was instituted to enable results to be analysed. Later it was found possible to differentiate between cases needing prisms to produce satisfactory results and those likely to be satisfied with sphero or sphero-cylindrical corrections without prisms. Decision on that point is now made as a result of observation of the balanced condition of the ciliary muscles, knowledge of latency, over stimulation of extrinsic muscles and visual habit factors.

The Analysis

Every case recorded and analyzed in plates one to three was a typical case of migraine. That is to say each one suffered from paroxysmal headaches preceded by scintillating scotoma. No cases of headache have been included in which the symptoms of scintillating scotoma were not present. The number of cases analyzed is 123. The columns show reading from left to right:—

(1) The indication number of the case in the analysis.
(2) The case record number.
(3) Whether male or female.
(4) Age.
(5) Frequency of attack.
(6) Position of headache.
(7) The prescription of lenses in use previous to examination and the visual acuity through those lenses or without lenses.
(8) Lenses prescribed after examination, also with visual acuity.
(9) Patient's report on examination after:—
A. four weeks. B. eight weeks. C. twelve weeks. D. six months and E. twelve months.

In the final column are notes referring to alterations of prescriptions given under column eight and which are recorded on the analysis sheet in plate 3. Certain diagnostic factors in connection with all the cases recorded on the analysis sheets have been omitted in the interests of a reasonable amount of simplicity in drawing up the sheets. For instance the type of person suffering from migraine is not noted but had the types been analyzed there would not have been any distinctive class predominating. Further migraine would have been shown not to have been more particularly associated with mental workers, than with any other type such as factory operatives, housewives or clerical workers. A review of the cases raises doubt as to any association between mental activity, manual work or any class of occupation and the cause of

migraine. The practice from which the cases have been collected is largely made up of working class people and tradesmen or their wives.

So far as could be ascertained the majority of the cases were, apart from migraine, healthy and free from disease or any chronic dysfunction, with the exception of digestive disorders and so-called "liverish" conditions. Digestive disorders were described by a large percentage of the cases. In all cases no ocular pathological conditions were discoverable.

While muscle balances, both lateral and vertical, were taken in all cases it cannot be found that any particular condition of 'phoria predominated. The variations from orthophoria would probably show a predominance in the direction of low exophoria as compared with esophoria but that would agree with the average found in a total of cases examined in ordinary practice. Many of the migraine patients showed slight hyperphorias, the majority of which were accommodative in origin. Very few cases have been noted in which there was a big departure from normality in lateral or vertical balance.

In many of the cases ductions were taken and the only point of importance noticeable is that they were somewhat low as compared with the average found in cases not migrainous. This refers both to abduction and adduction.

The numerical predominance of females over males is remarkable. Out of the total there are one hundred and five of the former and only eighteen of the latter, the proportion in percentage being 85% to 15%. To obtain true significance of these comparisons an analysis was made of the total number of cases examined in practice during the corresponding period covered by the analysis of migrainous cases. This was found to be 58% females and 42% males. Approximately this means that migraine in females predominates over migraine in males by four to one.

The analysis column four, given below, shows that there is no particular age associated with migraine. The fact that age forty-six shows the highest number of cases is of little significance when it is remembered that at about this age more people visit a refractionist than at other ages.

Age and Number of Cases

Female						Male					
Age	Cases	Age	Cases	Age	Cases	Age	Cases	Age	Cases	Age	Cases
15	2	32	5	46	8	18	1	29	3	41	1
16	2	33	2	47	1	20	1	30	1	44	1
17	2	34	1	48	2	22	1	34	1	45	1
18	4	35	5	50	2	26	2	35	2	65	1
20	2	36	3	52	1	27	1	39	1		
21	1	37	3	53	1						
22	6	38	2	58	1						
23	4	40	2	59	1						
26	6	41	2	61	2						
27	6	42	5	67	1						
29	6	43	2								
30	5	44	3								
31	2	45	2								

Case No.	Record No.	Male or Female	Age	Frequency of Attack	Position of Following Headache	WEARING										Add for Near		
						R					L							
						Sph.	Cyl.	Axis	△	V.A.	Sph.	Cyl.	Axis	△	V.A.	Sph.	△	J
1	N 36656	F	29	G	H.K.	−0·75	−1·25	180		·5	−1·50	−1·50	180		·4−1			
2	N 36787	F	27	F	H.		−1·0	100		1·	−0·25	−0·50	180		1·			
3	N 36812	F	32	F	H.K.	+0·50				1·	+0·50				1·			
4	N 36844	F	34	A	H.K.		+0·75	70		1·		+0·75	60		1·			
5	NCH 36673	M	26	F	H.K.			N W G		1·					1·			
6	NCH 36838	F	26	G	H.K.			N W G		1·					1·			
7	NCH 30897	M	29	G	H.K.	−1·50				1·	−1·50				1·			
8	NCH 36919	F	30	G	H.K.			N W G		1·−3					1·			
9	N 36929	F	27	G	H.K.			N W G		·33−1					·33−1			
10	N 36962	F	27	G	H.K.		+0·50	90		·5−1		+0·50	90		·5−2			
11	NCH 37012	F	17½	G	H.			N W G		·66−3					·66−3			
12	N 37068	F	23	B	H.K.		+0·50	70		1·		+0·50	90		1·			
13	NHI 37074	F	21	G	H.K.			N W G		1·					·1·			
14	NHI 37136	F	23	G	H.K.			N W G		1·					1·			
15	N 37152	F	37	F	H.	−0·75	−0·25	180		1·	−0·75	−0·25	180		1·			
16	NCH 37156	F	20	B	H.			N W G		1·					1·			
17	NCH 37192	F	31	F	H.	+3·0	−0·50	90		·66+2	+3·25	−1·25	60		·8			
18	N 37228	F	15	G	H.		−0·50	180		1·		−0·50	180		·66+			
19	NHI 37248	F	23	F	H.K.			N W G		1·−1					1·−1			
20	NHI 37333	F	20	G	H.K.			N W G		·66+2					·66+2			
21	N 37341	F	16	G	H.			N W G		·5−2					·4			
22	NCH 37347	F	29	G	H.			N W G		1·−2					1·−2			
23	NHI 37398	F	29	F	H.	−2·75	−0·50	60		1·	−2·75	−0·50	100		1·			
24	NCH 37425	M	30	G	H K L			N W G		·66					·66			
25	N 37480	F	48	G	H.L.			N W G		1·−1					1·−1			
26	N 37578	F	45	G	K.L.			N W G		·5					·66+2			
27	N 37584	F	17	G	H.			N W G		·66−2					·66−2			
28	N 37589	F	44	G	H. R.E.			N W G		·5					·5+2			
29	NHI 41547	F	46	G	K.L.			N W G		·4					·4			
30	NCH 41580	M	29	A	H.			N W G		1·					1·			
31	N 35427	M	41	A	H K L	pl				1·	+0·75	−0·75	25		1·			
32	N 41652	F	15	G	K.			N W G		·66−1					1·			
33	NCH 41701	F	37	G	H.			N W G		1·					1·			
34	N 41729	F	26	B	H.K.		−0·75	180		1·−1	−0·25	−0·75	180		·66+1			
35	N 41738	F	59	B	H.K.			N W G		·25					·2			
36	NCH 41758	F	22	A	H.K.	+0·75	+0·50	90		1·	+0·50	+0·75	105		1·			
37	N 41766	F	18	F	H.			N W G		·5					·5			
38	NCH 41771	F	18	F	H.	+1·0			1 in.	1·	+1·0			1 in.	1·			
39	N 41789	F	35	G	H.			N W G		1·					1·			
40	N 41794	M	45	G	H.	−1·0	−0·50	90		·4	−1·0	−0·75	90		·66			
41	N 41888	M	27	D	H.K.	+0·75	+0·25	90		1·	+0·50	+0·50	90		1·			
42	N 41924	F	32	F	H.K.			N W G		·5					·5			
43	NHI 41953	M	18	F	H.L.			N W G		·33					·5			
44	NHI 42002	F	38	G	H.K.			N W G		·5					·5			
45	N 42015	F	26	G	H.M.			N W G		·8					·8			
						Crookes												
46	N 42144	F	29	G	H.	−4·50	−0·50	20		·5	−4·25				·66+2			
47	NCH 42170	F	43	G	H.K.	−0·75	−3·0	135		·5	−0·75	−2·0	45		·8			
48	N 42428	F	18	G	L.M.			N W G		1·					1·			
49	NCH 42463	F	46	F	L.			N W G		·5+2					·4			
50	N 42523	F	16	F	H.			N W G		1·					1·			
51	NHI 42591	F	46	F	H.	+0·75	−1·0	180	1 in.	1·	+1·25	−1·0	175	1 in.	1·	+1·0	2 in.	1
52	N 42608	M	34	E	H.K.			N W G		·8					·8			
53	N 42621	F	41	F	H.K.			N W G		·8−1					·66−1			

Plate One

Case No.	PRESCRIPTION GIVEN										Add for Near			REPORTS					NOTES
	R					L								4 Weeks	8 Weeks	12 Weeks	6 Months	12 Months	
	Sph.	Cyl.	Axis	△	V.A.	Sph.	Cyl.	Axis	△	V.A.	Sph.	△	J						
1	−0·25	−2·0	180	3 in.		−1·25	−1·50	160	3 in.		+0·50		1				D	A	
2	+0·25	−1·0	105	1½ in.	1·	+0·25	−0·50	180	1½ in.	1·			1		A	A			See Re 1
3	+1·0			1 in.		+1·0			1 in.				1						
4	+1·0	−0·75	170	½ in.	1·	+1·25	−1·0	150	½ in.	1·			1			A			
5	+0·75	−0·50	180	1½ in.	1·	+1·0	−0·50	180	1½ in.	1·			1					C	
6		+0·50	95	½ in.	1·		+0·50	85	½ in.	1·			1	B					See Re 2
7	−1·25			1 in.	1·	−1·25			1 in.	1·			1			C	C	A	
8		+0·25	80	1 in.	1·		+0·25	100	1 in.	1·			1				A		See Re 3
9		−1·0	100	1 in.	1·	−0·25	−1·0	70	1 in.	1·			1				C		See Re 4
10	+1·50	−1·0	5	1 in.	1·−2	+1·25	−1·25	175	1 in.	1·−2			1		B	B			See Re 5
11		−0·50	90	1 in.	1·	+0·25	−0·25	90	1 in.	1·			1	A	A	A	A	A	
12		+0·75	80	1 in.	1·	+0·25	+0·50	100	1 in.	1·			1		C			A	
13	+0·25			1 in.	1·	+0·50			1 in.	1·			1		C	C			See Re 6
14		+0·75	100		1·	+0·25	+0·50	90		1·			1				C		
15	−0·50				1·	−0·50				1·			1						
16	+0·25	−0·75	175		1·	+0·50	−0·75	165		1·			1						
17	+4·50	−1·0	110		1·−2	+4·50	−1·50	70		1·			1				A		
18	+0·25	+0·50	90	1 in.	1·		+0·50	90	1 in.	1·			1			A			
19	+0·50			1 in.	1·	+0·50			1 in.	1·			1		A				
20	+0·50	−1·0	5		1·−1	+0·50	−1·0	165		1·−1			1			A			
21	+0·50			1 in.	1·−1	+0·50			1 in.	1·−1			1			A			See Re 7
22	+0·50	+0·25	30		1·	+0·50	+0·25	150		1·			1						
23	−2·25	−0·75	55	1 in.	1·	−2·25	−0·75	120	1 in.	1·			1						
24	+0·75	−0·50	170	1 in.	1·	+0·75	−0·50	180	1 in.	1·			1	A	A	A	A	A	
25		+0·50	80		1·		+0·50	30		1·	+0·75		1	A	A	A	A	A	
26	+1·25	−1·0	85		1·	+0·75				1·			1					B	
27	+1·0	−0·50	10		1·	+1·0	−0·50	165		1·			1			A			
28	+2·0				1·	+1·75				1·			1				C		See Re 8
29	+1·50				1·	+1·50				1·	+0·50		1						
30	+0·25	−0·25	180		1·	+0·75	−0·25	180		1·			1	A	A	A	A	A	
31	+0·50			1 in.	1·	+0·50	+0·75	100		1·	+0·50		1		A			A	
32	−0·50	−0·25	65		1·	Pl				1·			1			A			
33	Pl			1 in.	1·	Pl			1 in.	1·			1						
34	+0·75	−0·50	15		1·		+0·75	85		1·			1		A			A	See Re 9
35	+1·50	−0·50	110		1·	+1·75	−0·50	70		1·−1			1			A			
36	+1·25	−0·75	5	1 in.	1·	+1·50	−0·75	15	1 in.	1·			1					A	
37	−0·50				1·	−0·75				1·			1						
38	+0·50				1·	+0·50				1·			1			A			
39	+0·25	−0·50	165	1½ in.	1·	Pl			1½ in.	1·			1			B	B	A	See Re 10
40	−0·25	−1·25	105		·5	−0·25	−1·50	75		1·			1	A	A	A	A	A	
41	+1·0	+0·25	90	1 in.	1·	+1·0	+0·25	90	1 in.	1·			1			B			See Re 11
42	+0·25	−0·50	160		1·	+0·25	−0·50	20		1·			1			A			
43	−0·25	−0·75	100		1·		−0·75	80		1·			1						
44	+2·50	+0·50	180		1·	+2·25	+0·50	180		1·			1						
45	+0·50				1·	+0·50				1·			1			B		A	See Re 12
46	−4·25			1½ in.	·66	−4·0			1½ in.	·66			1	A					
47	−1·25	−2·75	135		·5	−2·0	−2·50	40	4 down	·5	+1·50		1	A					
48	+0·75			2 in.	1·	+0·50			2 in.	1·			1	A					
49	+0·50	−0·50	120		1·	+0·75	−1·0	55		1·			1						
50	+1·50				1·	+1·50				1·			1						
51	+0·75	−1·0	180		1·	+1·25	−1·0	175		1·	+0·75		1						
52		+0·25	35	1 down	1·		+0·25	145		1·			1				B		See Re 13
53		−0·25	100		1·	+0·25	−0·75	110		1·	+1·0		1			B			See Re 14

Plate One

Case. No.	Record No.	Male or Female	Age	Frequency of Attack	Position of Following Headache	WEARING										Add for Near		
						R					L							
						Sph.	Cyl.	Axis	△	V.A.	Sph.	Cyl.	Axis	△	V.A.	Sph.	△	J
54	NCH 42650	F	22	G	H.K. R. half of head	+1·25				·66−2	+1·25				·8−2			
55	NCH 42700	F	46	G	H.K.			N W G		·8−3					·8−3			
56	NCH 42819	F	45	G	H.K.			N W G		·8−1					·8−1			
57	NCH 42828	F	35	G	H.K.			N W G		·8−1					·8−1			
58	NHI 42829	F	32	F	H.		+0·25	90		1·		+0·25	90		1·			
59	N 42873	F	46	G	H.K.	−2·50	−2·0	180		·8	−3·0	−1·50	180		·8			
60	N 42874	F	44½	B	H.			N W G		·66−2					·66−2			
61	N 43004	F	22	G	H.			N W G		1·					1·			
62	NHI 43087	F	29	G	H.K.	+0·75	+0·25	90		1·	+0·75				1·			
63	NHI 43111	F	18	A	H.K.			N W G		·8−1					·25			
64	NHI 43212	F	67	F	H.K.	+3·0	−0·25	135		·8−3	+3·0	−0·50	25		·8−3	+2·50		1
65	NHI 43224	M	35	F	H.			N W G		·66+2					·5			
66	NCH 43248	F	42½	F	H.L.			N W G		·8−1					·8−1			
67	NCH 43274	F	61	G	H.K.	+7·0	+1·0	90		·2	+5·50	+0·50	90		·4−1	+2·50		1
68	NCH 43300	F	41	G	H.K.	+0·50				·66+2		+0·50	90		·66			
69	NCH 43311	F	42	G	H.	−0·25	−0·25	180		1·	−0·25	−0·25	90		1·			
70	N 43307	F	35	G	H.K.			N W G		1·					1·			
71	N 43326	F	35	G	H.	−16·0	−3·0	150		·25+1	−9·0	−5·0	30		·2			
72	N 43378	F	42	G	H.			N W G		1·					1·			
73	N 43470	F	30	F	H.		+0·50	180		·5+1		+0·25	180		·5+1			
74	N 43526	F	46	A	K.L.			N W G		·3					·3			
75	N 43531	F	37	F	H.		+0·75	90		·5		+0·75	90		·5			
76	N 43568	F	36	F	H.	+2·0	−1·0	105		·5−2	+2·50	−1·0	30		·25−1			
77	N 43578	F	23	F	H.K.			N W G		1·					1·			
78	N 43592	F	33	A	H.		+0·25	180		·66−2		+0·25	180		·66			
79	N 43601	F	58	E	H.			N W G		·2					·2			
80	N 43617	F	27	B	H.K.	+0·50	−0·25	90		·8+		+0·25	180		·8+1			
81	NCH 43623	F	31	G	H.K.	−1·0	−1·0	125		·4+2	−1·0	−1·0	80		·5+1			
82	N 43633	F	30	G	K.	+0·25	+0·25	180		·8	+0·50				·8			
83	N 43647	F	46	A	H.	+0·25	−0·75	155		·5	+0·25	−0·75	10		·5			
84	N 43664	F	46	A	H.		+0·50	25		1·		+0·25	135		1·			
85	N 43681	F	42	A	K.	−0·75	−0·50	180		·66	−0·75	−0·25	180		·25			
86	N 43684	F	47	A	H.L.			N W G		·8−2					·5−1			
87	N 43689	F	38	A	H.L.	+0·50	−1·75	135		·66−2		−2·75	45		·06			
88	N 43711	F	22	A	H.L.	−3·0				·5	−3·0				·14			
89	N 43744	F	43	G	HLK	+0·75	+0·75	135		1·	+0·25	+0·25	180		1·			
90	N 43769	F	48	A	H.K.	−0·25				·66+1	−0·25				1·	+1·75		1
91	N 43708	M	22	G	H.			N W G		1·					1·−2			
92	NCH 41136	F	49½	G	H.K.	+1·50	+0·50	180		1·	+1·25	+0·50	180		1·			
93	NCH 40969	F	36	A	H.K.	+0·50	+0·75	90		·8−3	+0·25	+0·75	90		·8−1			
94	N 40271	F	26	G	H.K.		+3·50	80		·5		+2·50	95		·5+1			
95	N 40898	M	65	G	H.	+0·75	+0·50	165		·5	+0·75	+0·50	15		·66+3			
										Uncorrected					Uncorrected			
96	NHI 41314	F	20	E	H.	+1·0				·5−2	+1·0				·5−2			
97	N 43372	F	42	F	H.			N W G		·66					·66			
98	N 41397	F	40	A	H.K.	+1·0	−1·50	175		·8	+1·0	−1·50	10		·8			
99	N 41448	F	44	B	H.K.	−0·75				·66−3	−0·75				·66−3			
100	N 41483	F	22	C	H.K.	+0·75				1·−1	+1·50				1·			
101	NCH 43576	M	44½	A	H.K.		+0·50	90		·4	+0·75	+0·25	90		1·−2			
102	N 43895	M	29	A	H.K.			N W G		·2					·33+2			
103	N 43699	F	61	G	H.	Pl					+2·50				·5			
104	N 43751	F	35	G	H.			N W G		·5+2					· 5			

Plate Two

Case No.	PRESCRIPTION GIVEN										Add for Near			REPORTS					NOTES
	R					L								4 Weeks	8 Weeks	12 Weeks	6 Months	12 Months	
	Sph.	Cyl.	Axis	△	V.A.	Sph.	Cyl.	Axis	△	V.A.	Sph.	△	J						
54	+1·50	−1·0	90		1·	+1·50	−0·50	90		1·			1						
55	+1·0					+0·75							1				A		
56		+0·25	90		1·	+0·75				1·			1					F	
57		−0·25	95	1½ in.	1·	+0·25	−0·25	45	1½ in.	1·			1	B			B	A	
58	+0·25	−0·25	45	1 in.	1·	+0·25	−0·25	180	1 in.	1·			1	A					
59	−2·75	−1·75	180	2½ in.	1·	−2·50	−1·0	5	2½ in.	1·			1						
60	+0·75				1·	+0·50				1·			1						
61	+0·50				1·	+0·75				1·			1			A	A		See Re 15
62	+1·25	−0·25	20	1 in.	1·	+1·0	−0·37	15	1 in.	1·			1		A		A		
63	+3·50	−0·75	140		1·	+3·75	−0·75	175		1·			1		A				See Re 20
64	+3·0			1 in.	·8	+3·0	−0·50	180	1 in.	·8	+2·50	4 in.	1	A	A	A	A	A	
65		−0·50	70	1 in.	1·	−0·50	−0·25	15	1 in.	1·			1						
66	+0·75	−0·50	55	1½ in.	1·	+0·75	−0·25	180	1½ in.	1·	+2·0	3 in.	1						
67	+7·0	+1·0	90			+6·0	+0·50	90		·66	+3·0		1				A		
68	+0·50	−0·25	140	1 in.	1·	+0·75	−0·75	20	1 in.	1·			1			A	A		
69	+0·25	−0·50	180	2 in.	1·	−0·25	−0·25	105	2 in.	1·			1	A					
70	Pl				1·	+0·50	−0·25	135		1·			1	B					See Re 16
71	−18·0	−2·0	165	3 in.	·4+2	−12·0	−3·50	30	2 down	1·			1				C		
72	+0·50	−0·25	170	1 in.	1·	+0·50			1 in.	1·			1						
73	+0·75	−1·0	90	1 in.	1·	+0·50	−0·25	90	1 in.	1·			1						
74	+0·75	−0·75	90		1·	+0·75	−0·50	90		1·	+0·75		1			A			
75	+1·25	−0·75	180	1 in.	1·	+1·0	−0·75	180	1 in.	1·			1			A			See Re 17
76	+1·75	−1·50	120		1·	+2·75	−1·50	30		1·			1						
77	+0·25	−0·50	20	1 in.	1·	+0·25	−0·50	180	1 in.	1·			1						
78		+0·25	175	1½ in.	1·		+0·25	135	1½ in.	1·			1				B		
79	+1·0	−0·25	90		1·	+1·25	−0·25	20		1·			1			B			
80	+0·50	−0·25	105	1 in.	1·	+0·25	−0·37	45	1 in.	1·			1		A				
81	−0·75	−1·0	90	2 in.	·66−2	−0·75	−1·0	90	2 in.	·66−2			1						
82		+0·37	95	1½ in.	1·	+0·25	+0·25	90	1½ in.	1·			1	A					
83	+0·25	−0·50	150	1 in.	·8		−0·50	10	1 in.	·8	+0·75	2 in.	1			B			
84	+1·0	−0·50	125		1·	+1·0	−0·50	50		1·	+0·75		1			A			
85	−0·25	−0·50	175	½ in.	·8	−1·0	−0·50	180	½ in.	·5	+1·50	1 in.	1		A				
86	+0·25	−0·50	35	1½ in.	1·	−0·25	−0·25	180	1½ in'	1·−2 less	+0·75	3 in.	1		C				See Re 18
87	+0·50	−1·50	130	1 in.	1·	+1·0	−2·50	55	1 in.	·06			1		A				
88	−2·50	−1·0	15	1 in.	·5	−2·50	−1·0	175	1 in.	·5			1		B				
89	+0·75	+0·75	135	1 in.	1·	+0·50			1 in.	1·			1			B			
90	+1·0	−0·25	130	1 in.	1·	+0·75	−0·25	20	1 in.	1·			1						
91	+1·25	−0·50	170	1 in.	1·	+1·75			1 in.	1·			1		C				
92	+0·25	+0·50	180	1½ in.	1·		+0·50	180	1½ in.	1·	+1·50		1		B		A		
93	+1·0			1½ in.	1·	+1·0	−0·50	180	1½ in.	1·			1						See Re 10
94		+3·0	90		·5		+2·0	100		·5			1					A	
95	+1·0	+0·50	165	3 in.	·66	+0·75	+0·50	15		1·	+2·50		1						
96		−0·75	170	1 in.	1·	−0·25	−0·25	180	1 in.	1·			1		A				
97	+1·0	−0·50	180		1·	+0·50	−0·25	180		1·			1			B			
98	+1·25	−1·75	175	1 in.	1·	+1·25	−2·0	7½	1 in.	1·			1		B		C		
99	−0·25	−0·25	100	1 in.	·8	−0·25	−0·25	90	1 in.	1·			1			A			
100	+1·50			1 in.	1·	+1·50			1 in.	1·			1		A				
101	+1·0	−0·25	140	1 in.	·5+1	+0·75	−0·25	180	2 up	1·			1			C			See Re 21
102	+0·75	−0·75	10	1 in.		+0·75	−0·75	180		·66 bin oc.			1	A					
103	Pl					+0·25	−1·0	180		1·	+2·50		1		B				
104	−0·25			1 in.	1·	Pl			1 in.	·66			1		A				

Plate Two

Case No.	Record No.	Male or Female	Age	Frequency of Attack	Position of Following Headache	WEARING R Sph.	R Cyl.	R Axis	R △	R V.A.	WEARING L Sph.	L Cyl.	L Axis	L △	L V.A.	Add for Near Sph.	Add for Near △	Add for Near J
105	N 42144	F	29	G	H.	−4·50	−0·50	20		·5−1	−4·25				·66+2			
106	N 37387	M	35	G	H.K.			N W G		1·					1·			
107	N 40200	M	20	G	H.K.	−2·50	−0·50	90		1·	−1·0	−0·25	90		1·			
108	N 41367	F	26	A	H.		+0·50	10		·4−1	+0·75	−0·75	55		·6−2			
109	N 41558	F	50	G	K.H.	−3·75				·8	−3·50				·8−1			
110	N 41527	F	53	B	H.K.	+1·25					+1·25							
111	NH1 43877	F	27	B	H.K.	+0·25	+0·50	90		1·−2	+0·50	−0·75	180		1·−2			
112	N 42915	F	30	B	H.	−0·50				·66+3	−0·50				·66−3			
113	N 41595	F	33	G	H.L.	+1·50	−0·50	85		·8+	+1·25	−0·25	30		1·			
114	NCH 43889	F	27	C	H.			N W G		1·					1·			
115	NCH 43986	F	32	A	H.			N W G		1·					1·			
116	N 43894	F	52	G	K.					1·					1·			
117	N 44041	F	36	A	H.K.	+1·0				1·	+1·0				1·			
118	N 41236	F	32	B	H.	+0·75	−0·75	150		1·	+0·75	−0·25	165		1·			
119	N 41284	F	40	G	H.K.	+0·25	−0·50	90		1·	+0·25	−0·75	95		·4+			
120	N 35431	F	30	A	HJL			N W G		1·					1·			
121	N 39632	F	22	A	HKL	−4·50	−2·50	105		·5	−2·50	−2·25	65		·5			
122	N 35379	M	26	A	K.L.	−1·75	−0·50	180		1·	−0·25				1·			
123	N 36759	M	39	G	H.K.	−1·50	−3·25	7½		·8+1	−1·50	−2·75	180		·8+2			

RETESTS.

Case No.	Record No.	Male or Female	Age	Frequency of Attack	Position of Following Headache	WEARING R Sph.	R Cyl.	R Axis	R △	R V.A.	WEARING L Sph.	L Cyl.	L Axis	L △	L V.A.	Add for Near Sph.	Add for Near △	Add for Near J
1	N 39726	F	27			+0·25	−1·0	105	1½ in.	1·	+0·25	−0·50	180	1½ in.	1·			1
2	N 39631	F	26				+0·50	95	½ in.	1·		+0·50	85	½ in.	1·			1
3	N 39965	F	30				+0·25	80	1 in.	1·		+0·25	100	1 in.	1·			1
4	N 39900	F	27				−1·0	100	1 in.	1·−2	−0·25	−1·0	70	1 in.	1·			1
5	N 39760	F	27			+1·50	−1·0	5	1 in.	1·	+1·25	−1·25	175	1 in.	1·			1
6	N 39889	F	21			+0·25			1 in.	1·	+0·50			1 in.	1·			1
7	N 40349	F	16			+0·50			1 in.	1·−1	+0·50			1 in.	1·−1			1
8	N 40391	F	44			+2·0				1·−1	+1·75				1·−1			1
9	N 40835	F	27			+0·75	−0·50	15		1·		+0·75	85		1·			1
10	N 40439	F	35			+0·25	−0·50	165	1½ in.	1·	Pl			1½ in.	1·			1
11	N 40346	M	27			+1·0	+0·25	90	1 in.	1·	+1·0	+0·25	90	1 in.	1·			1
12	N 40627	F	26			+0·50				1·	+0·50				1·			1
13	N 41105	M	34				+0·25	35	1 down	1·		+0·25	145		1·			1
14	N 40726	F	41				−0·25	100		1·	+0·25	−0·75	110		1·	+1·0		1
15	N 41443	F	22															1
16	N 41180	F	35			Pl				1·	+0·50	−0·25	135		1·			1
17	N 41610	F	37			+1·25	−0·75	180	1 in.	1·	+1·0	−0·75	180	1 in.	1·			1
18	N 41685	F	47			+0·25	−0·50	180	1½ in.	1·	−0·25	−0·25	180	1½ in.	1·−2	+0 75	3 in.	1
19	N 41141	F	36			+1·0			1½ in.	1·	+0·50	+0·50	90	1½ in.	1·	+1·50		1
20	N 41158	F	18			+3·50	−0·75	140		1·	+3·75	−0·75	175		1·			1
21	N 41605	M	44½			+1·0	−0·25	140	1 in.	·5+1	+0·75	−0·25	180	2 up	1·			1
22	N 41627	F	53			Pl			1 in.	1·—	Pl			1 in.	1·−	+1·25		1
23	N 41777	F	30			Pl			1 in.	·66+3	Pl			1 in.	·66+3			1
24	N 39569	F	30			+0·50			1 in.	1·	+0·50			1 in.	1·			1
25	N 40114	F	22			−3·50	−2·75	100	1½ in.	·5	−1·75	−2·50	70	1½ in.	·5			1
26	N 41140	M	26			−1·37	−0·50	170	1½ in.	1·	+0·87			1½ in.	1·	+0·50		1

Plate Three

Case No.	PRESCRIPTION GIVEN										Add for Near			REPORTS					NOTES
	R					L								4 Weeks	8 Weeks	12 Weeks	6 Months	12 Months	
	Sph.	Cyl.	Axis	△	V.A.	Sph.	Cyl.	Axis	△	V.A.	Sph.	△	J						
105	−4·25			1½ in.	·8−1	−4·0			1½ in.	·8−1	+0·50		1	A		A			
106	+1·0	−0·50	180	1 in.	1·	+0·75			1 in.	1·			1						
107	−2·50	−0·50	90	1 in.	1·	−1·0	−0·25	90	1 in.	1·			1		A				
108	+0·75	−0·50	100	1 in.	1·−1	+0·75	−0·50	55	1 in.	1·			1		C	A			
109	−3·50	−0·25	90	1 in.	·8	−3·25	−0·25	30	1 in.	·8	+1·50		1		B				
110	Pl			1 in.	1·−1	Pl			1 in.	1·−1	+1·25		1		A				See Re 22
111	+1·0	−0·75	180	1 in.	1·−2	+1·25	−1·25	175	1 in.	1·−2			1			A			
112	Pl			1 in.	·66+3	Pl			1 in.	·66+3			1		A				See Re 23
113	+1·50	−0·50	85	¾ in.	1·	+1·25	−0·25	30	¾ in.	1·			1			A			
114	+0·50	−0·50	95	1 in.	1·	+0·75	−0·75	85	1 in.	1·			1			A			
115	+0·50			1 in.	1·	+0·50			1 in.	1·			1		A				
116	Pl			1 in.	1·	Pl			1 in.	1·	+1·75		1			A			
117	+1·0	−0·50	130	1 in.	1·	+0·50	−0·50	20	1 in.	1·			1		A				
118	+0·75	−0·75	150	1 in.	1·	+0·75	−0·25	180	1 in.	1·			1					A	
119	+0·75	−0·50	95	1½ in.	1·	+1·0	−1·0	95	1½ in.	1·			1		A				
120	+0·50			1 in.	1·	+0·50			1 in.	1·			1				B		See Re 24
121	−3·50	−2·75	100	1½ in.	·5	−1·75	−2·50	70	1½ in.	·5			1				B		See Re 25
122	−1·37	−0·50	170	1½ in.	1·	+0·87			1½ in.	1·	+0·50		1					A	See Re 26
123	−1·25	−3·50	12½	3 in.	1·	−1·0	−2·50	177½	3 in.	1·			1					A	

RETESTS.

Case No.	PRESCRIPTION GIVEN										Add for Near			REPORTS					NOTES
	R					L								4 Weeks	8 Weeks	12 Weeks	6 Months	12 Months	
	Sph.	Cyl.	Axis	△	V.A.	Sph.	Cyl.	Axis	△	V.A.	Sph.	△	J						
1		−1·0	105	1½ in.	1·		−0·50	180	1½ in.	1·			1				A		
2	+0·25	+0·50	95	1 in.	1·	+0·25	+0·50	85	1 in.	1·			1			A			
3	+0·50			1½ in.	1·	+0·75			1½ in.	1·			1					A	
4		−1·0	100	1 in.	1·		−1·0	70	1 in.	1·	+0·50	2 in.	1						
5	+1·75	−1·0	5	1 in.	1·	+1·50	−1·25	175	1 in.	1·			1		A				
6	+0·50			1 in.	1·	+0·50			1 in.	1·			1					A	
7	+0·75			1 in.	1·	+1·0			1 in.	1·			1					A	
8	+2·50				1·	+2·25				1·			1						
9	+0·75	−0·25	180		1·	+0·75	−0·50	180		1·			1					A	
10	+0·75	−0·50	165	1½ in.	1·	+0·50			1½ in.	1·			1				A		
11	+1·0	−0·25	180	1½ in.	1·	+1·50	−0·25	180	1½ in.	1·			1					A	
12	+0·50			1 in.	1·	+0·50			1 in.	1·			1			A			
13	+0·50	−0·25	125		1·	+0·50			1 up	1·			1						
14		−0·25	100	1½ in.	1·	+0·25	−0·75	110	1½ in.	1·	+1·0		1					A	
15													1					A	
16	Pl			1 in.	1·	+0·50	−0·25	135	1 in.	1·			1	A					
17	+1·50	−0·75	180	1½ in.	1·	+1·25	−0·75	5	1½ in.	1·			1					A	
18		−0·50	25	1½ in.	1·	−0·25	−0·25	180	1½ in.	1·	+1·25	3 in.	1					A	
19	+1·25			2 in.	1·	+1·50	−0·50	180	2 in.	1·			1					A	
20	+4·0	−0·75	140		1·	+4·25	−0·75	175		1·			1					A	
21	+1·0	−0·25	140	2 in.	1·	+0·75	−0·25	180	2 up	1·			1					A	
22	−0·25			1 in.	1·	−0·25			1 in.	1·			1					A	
23	−0·25	−0·25	125	1 in.	1·		−0·25	90	1 in.	1·	+1·0	2 in.	1					A	
24	+1·0			1½ in.	1·	+1·0			1½ in.	1·	+0·25	5 in.	1				A		
25	−3·75	−2·50	100	3 in.	·5	−2·0	−2·50	70	3 in.	·5			1	A					
26	−2·50	−0·25	180	2 in.	1·		+0·25	90	2 in.	1·	+0·50	4 in.	1					A	

Plate Three

From column five the following Table of frequency of attack has been made. The letter preceding the period mentioned is the key letter given on the analysis sheets:—

Female—105 *Cases*		*Male*—18 *Cases*	
A. Daily	19	A. Daily	5
B. Bi-weekly	11	D. Fortnightly	1
C. Weekly	2	E. Monthly	1
D. Fortnightly	0	F. Occasional	3
E. Monthly	2	G. Frequently	8
F. Occasional	21		
G. Frequently	50		

The word "frequently" (G) was used in connection with cases who could not state a definite period between attacks. Many will tell of three or four attacks within a week followed by a fortnight's respite or even longer. Under this heading are cases who looked upon themselves as liable to an attack at anytime and who had a sufficient number of attacks to live in more or less dread of one occurring. It will be noted that a high percentage suffered daily. A few included under the heading of "daily" did not have an attack on Sunday. Under E (Female list) the smallness of the number of cases (two only) is significant in view of the suggested association of menstruation and migraine. Consideration of the high number under A and G, both female and male, is significant. A high percentage of cases, obviously, must live and work in constant dread of an attack and be greatly incapacitated by their sufferings especially when the mental distress associated with migraine is realized.

The Key to the analysis of Position of Headache is as follows:—

Frontal	H.
Temporal	K.
Vertical	J.
Occipital	L.
Supra-orbital	M.

The analysis is as follows:—

Females		*Males*	
H. K.	44	H. K.	9
H.	41	H.	5
H. K. L.	2	H. L.	1
L.	1	H. K. L.	2
H. L.	6	K. L.	1
L. M.	1		
K. L.	3		
H. M.	1		
K.	5		
H. J. L.	1		

It will be seen, from further extractions, that frontal headaches are present in 111 out of the 123 cases, although only forty-six are recorded as frontal only. Temporal headaches were noted in sixty-five

cases but that headaches in other parts of the head were also present is shown by the fact that five only were entirely restricted to the temporal region. It will be remembered that the diagnosis of migraine was based, essentially, upon the presence of scintillating scotoma as well as paroxysmal headache. Frequently the description of headache would be "commences in the temples but soon spreads to the forehead."

From column seven, the record of lenses in use before examination, it is shown that the total number of migraine cases who had worn a correction and continued to suffer from migraine was sixty-seven. The number not having worn a previous correction was fifty-six.

The visual acuity before examination is shown in the following Table:—

Each eye—1·0	62
Each eye—0·8 or 0·6	18
Each eye—0·5	15
Each eye—less than 0·5	13
Not less than 0·5 in either eye	8
Less than 0·5 in one eye and better than 0·5 in the other eye	7

From this Table it will be seen that the majority of migraine cases have good visual acuity This supports any suggestion that attacks may be caused, or precipitated, by auto-correction of refractive errors. Out of the one hundred and twenty-three cases only seven were found not to have demonstrable errors of refraction. Therefore auto-correction of the refractive errors was habitual and carried to the extent approximating normal visual acuity as far as possible.

An analysis of column eight (prescriptions given) is as follows:—

Minus Spheres	6
Minus Sphero-cylinders	19
Plus Spheres	13
Plus Sphero-cylinders	60
*Mixed Astigmatism	15
Plus one eye and minus the other eye	3
Reading correction only	3
Plano-prisms	4

Approximately half of the corrections were under one dioptre; a quarter of the total were between one and two dioptres, the remainder were over two dioptres. In arriving at the figures the strength of the sphere has been added to the strength of the cylinder. There is nothing in the Table to suggest that any particular error of refraction is more liable to cause migraine. It rather suggests that any error may be the cause or precipitating factor.

An analysis of the reports given subsequently by the cases after use of the lenses prescribed is as follows:

A. Complete relief	69
B. Scintillations and Headaches less frequent and severe	17

C. Isolated attack in the period under review	11
Not reported	26
	123

Some of the cases tabulated under B and C had an alteration of lenses at the time the first report was received. Such alterations are shown on the analysis sheet plate 3. It will be noted that all reports in plate 3 are under the key letter A (complete relief).

Prisms were included in the prescriptions of eighty cases out of the total of one hundred and twenty-three analyzed: seventy-nine were given base in, the odd one being vertical. Their use, at first, was necessarily experimental and the amount given somewhat empirical. It was found that most cases could wear base in prisms equivalent to one third of the amount of the distance abduction recovery (the prism through which resumption of single vision is obtained following the "break" in taking total abduction measurement), without difficulty. As the average recovery point was six prism dioptres, two prism dioptres became the average amount prescribed.

Many migraine cases, during a subjective test for ametropic corrections, present difficulties. This is noticed particularly when arriving at the amount of astigmatism present and the axis of the cylinder. The spherical element is also doubtful on many occasions. Visual fatigue becomes evident during examination. Small degrees of hyperphoria and, occasionally cyclophoria, are found. The hyperphoria varies in amount when measured through different test lenses. The addition of an empirically chosen pair of plus spheres added to the best distance correction found, will frequently eliminate any hyperphoria found at reading distance. Near point monocular tests and near point binocular visual and accommodative tests, frequently disclose unequal ciliary activity, unequal, that is, comparing one eye with the other. The differences may be small but are significant of imbalance. Esophoria, small in amount, at reading distance, is found in a number of cases, and is usually eliminated by plus spherical additions to a conventionally found correction. Dynamic retinoscopy will often disclose the need for more plus sphere to be added to one eye than to the other when the ciliary muscles are forced to relax to the utmost degree. Ductions are often low, both as regards abduction and adduction.

All these points indicate latent errors of refraction and, more often than not, unequal latent errors. So often has inequality of ciliary activity been noticed, an inequality presumably covering more refractive error in one eye than the other, that it has become an early point of investigation in all cases possessing symptoms of migraine as well as in many other cases of ocular distress and difficulty. The highest amount of difference of *latency* so far found has been 1·5 dioptres. It is rarely more than 0·75 dioptres. An amount of 0·25 dioptres has been found of great importance in many cases. It appears that *ciliary imbalance* covering unequal errors

of refraction, results in varying forms of extrinsic muscular imbalance which tend to disappear when the ciliary muscles are relieved of the necessity for unequal activity. The prescription of prisms base-in is in effect similar to the prescription of additional plus spheres when the ciliary muscles are considered The re-actions are in the directions of less expenditure of energy and the moving of the balance (or imbalance as the case may be) to a lower plane of stress. Prisms are indicated, at least as a temporary measure, in cases possessing any or all of the symptoms or conditions already mentioned which are indicative of latent errors, particularly unequal ones, and when the latency has become sufficiently fixed and habitual not to relax to a nearer condition of normality under the influence of spheres or sphero-cylinders. The prisms would not be considered necessary when, with the spherical or sphero-cylindrical elements in place, the ocular balance, refractively, accommodatively and muscularly proved to be normal, particularly upon examination at reading distance.

Owing to habit factors many small amounts of both equal and unequal latency cannot be given consideration in making up a prescription for constant wear without inconvenience and possible discomfort to the patient. In such cases the inclusion of the prisms has the desired immediate effect, and subsequently it may become possible to vary the spherical and sphero-cylindrical corrections and finally eliminate the prisms although this may not be possible for a long time perhaps, even for months or years. Whether the prisms are removable or not does not matter very much so long as the patient remains visually comfortable and free from the migraine headaches. No detrimental effect has yet been found by their use in the class of case under consideration.

A suitable procedure in examining a typical case of migraine would be: take History and Symptoms: record Visual acuity, monocular and binocular with old correction (if worn) or without lenses: take distance and near point 'phoria, lateral and vertical; perform distance retinoscopy and conventional subjective test for distance; dynamic retinoscopy; with distance correction already found in place and with a near point chart at forty centimetres find, monocularly, the strongest plus sphere which does not blur small type. Note any difference in the added sphere over each eye and compare with dynamic retinoscopic findings. An attempt should be made to add part or all of the *difference* to the original findings in the distance, adding to the one eye only but with both eyes open. Any such addition which does not give a suspicion of binocular blur can usually be worn and is indicated. The 'phorias should then be measured again and compared with those taken previously. The discovery of reduced esophoria, or reduced hyperphoria, but with some remaining, indicates the probability of reduced but incomplete reduction of ciliary imbalance, in which case one should add, empirically, weak plus spheres, either eye or binocularly, and note the effect upon the 'phorias. Having found a correction which gives the nearest approximation to normality of balance

at reading distance, a return should be made to the distance charts and the correction in the light of the findings at the near point carefully checked over. Some modification of the original distance correction may be possible in the direction indicated by the near point findings. If the distance and the near point balances (the word "balance" being used in the full sense) are good with the final correction found, prisms may be left out of the prescription. If there is doubt or a definite imbalance remains, prisms may be included in the prescription, after taking the ductions. Prisms are also indicated if the cylindrical element or axis is felt to be doubtful and also if, using the Jackson Crossed cylinder, a different cylinder at near is found when compared with that found in the distance (after the latter has been checked in the light of the difference found). The objects to be aimed at are clear binocular distance vision, balanced distance and near point visual acuity, balanced ciliary activity, normal extrinsic muscle balance, horizontal and vertical, and freedom from cyclophoria. These are not always obtained during the first examination and it is in the consideration of the nearness of approximation to them which guides one to the decision as to whether prisms are to be included or not.

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OSMOTIC PHENOMENA IN RELATION TO GLAUCOMA*

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It has been long accepted that the cornea is impermeable to liquids, whether from within out or from without in. Indeed, it is stated that the cells of the Substantia Propria may be described as occupying part of a regular system of cell spaces and canaliculi, but more recent investigations seem to indicate that during life they fill out the spaces completely, and leave no gaps through which fluids can pass. But: occasionally, leucocytes (wandering cells) are found between the fibrous elecents. (1) So much for the anatomical facts.

Inasmuch as we must herein consider Glaucoma as an anamoly of physiologic processes, let us investigate the alleged impermeability of the cornea, considering it as a membrane, and studying it in life.

The standard laboratory experiment for the demonstration of the permeability of a water-tight membrane is that of dialysis and osmosis. In this experiment in dialysis, we fill a small jar with salt or other crystalloid substance, cover it with a water-tight membrane, place it in a larger jar of distilled water, and discover that diffusion has taken place: an interchange of liquids *through* the *membrane*. If, further, we mix the salt in the smaller jar with a colloid (e.g., egg albumin), the salt will diffuse with the water as before, but the colloid will remain behind. "During (this) process of dialysis considerable pressure is developed, so much in fact that the membrane will very often give way and leak."

Pfeffer's experiments showed that the pressure developed within the small jar as above described, containing a six per cent solution of sugar, was sufficient to lift a column of mercury over ten feet. This pressure depends partly upon the strength of the crystalloid within the small jar, and to the fact that *this crystalloid does not diffuse outwards as rapidly as the water permeates in.*

Stated in brief, if water and a crystalloid be separated by a membrane, or thin tissue wall, diffusion will occur, the rate of diffusion being faster from the water into the crystalloid than from the crystalloid to the water, and osmotic pressure will develop within the container holding the crystalloid.

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We have seen that the cornea is considered to be a water-tight membrane in life. We can accept this in the same sense that we would *select* a water-tight membrane for experimentation in dialysis, and yet refuse to concede that it would not, under the circumstances allowed in experiments upon osmotic phenomena, act as any other membrane under similar circumstances.

We will, for example, consider the action of cyclopegics and mydriatics upon the sphincter pupillae. Atropine and homatropine are said to paralyze the third nerve endings in the sphincter, while eserine stimulates third nerve endings. Further, and more to the point, "one of these drugs is placed in the conjunctival sac, and from here it travels, *probably by diffusion* (author's italics) via cornea and aqueous, to the ciliary and iris muscles, both of which are paralyzed. *This fact is of the greatest importance, for if this path did not exist, the use of these valuable drugs would hardly be possible.*" (3) (Fuchs found that if after one eye had been dialated with atropine part of the aqueous of that eye were removed and placed in the conjunctival sac of the other eye that dialation would occur. Evidently the path *does* exist!)

That little word "diffusion" is the keystone of a new theory of the etiology of Glaucoma. We have seen what diffusion is. We have seen that when diffusion occurs between liquids of unequal densities that pressure of a considerable amount develops on that side of the membrane of the denser liquid.

It is known that in Glaucoma, one of the anatomical changes in the chemistry of the aqueous is that it becomes considerably more albuminous, so much so that it coagulates in the presence of air, or in hardening fluids. (4) It is also known that Glaucoma is accompanied or preceded by other senile changes such as fatty degeneration (arterio-sclerosis), and that in any degenerative process we will find that various salts, acids, glycerines and other alcohols are given off. (5)

It is therefore submitted that the cornea is physiologically permeable under certain chemical upsets of the aqueous, and that the collection of organic substances within the aqueous chamber not only acts as debris and as blockage agents within the spaces of Fontana, but that this blockage is *secondary* to an increase of intraocular pressure brought about by diffusion of lachrymal fluid within, at a greater rate than the albuminous aqueous diffuses *out* through the cornea (diosmosis).

We have the conditions necessary for an increase of intraocular pressure (which we assume by now to be osmotic in character) the moment the aqueous becomes albuminous through some chemical upset: a separating water-tight membrane having on the one side an albuminous (colloidal) compound, and on the other a (nearly) aqueous fluid. (The lachrymal fluid is actually very nearly a normal salt solution.) If, as Starling assumes, diffusion occurs through the cornea under certain conditions, it seems only logical to assume that it can and does occur in

the eye under the above nearly ideal laboratory conditions for the demonstration of increased pressure following dialysis, this pressure being termed osmotic.

If we assume that the above theory as to the cause of the increase of intraocular pressure in Glaucoma be true, in what manner, it may be asked, could we avoid, or treat such an increase of pressure, once it had started. Under laboratory conditions, we would place another crystalloid substance on the less dense or more nearly aqueous side of the membrane in order that diffusion would be more nearly equal in each direction—in such manner that diffusion would still occur, but with less great acceleration in the one direction than in the other, and with a consequent lowering of such pressure as may already have developed.

Lewis ([6]) reports repeated and almost invariable success in treating nearly hopeless cases of Glaucoma by using a solution of one per cent each of sodium bicarbonate, sodium chloride, and sodium biborate in a base consisting of equal parts of water and glycerine, followed immediately by exposure to the heat of a carbon-filament (infra-red) lamp at about 125° F. for daily periods varying from one-half to one hour. In one of his cases he reports a very steamy, oedematous cornea with only the margins of the iris to be seen, and the eye was intensely hard. He states that under the above outlined treatment that the corneal deposit "was seen to melt away as snow would under the sun," finally becoming perfectly clear, with all symptoms of Glaucoma and an accompanying uveitis disappearing finally and permanently.

He assumes that these results were due to the "dehydrating" action of the alkalines and of the heat. In the writer's opinion, as suggested above, osmotic pressure is equalized on both sides of the cornea, the aqueous made less dense with the infiltrated lachrymal fluid, allowing it to pass off with greater ease through the normal channels, thus releiving the Glaucomatous symptoms; that, further, the deposits within the cornea are carried off during the process of dialysis, in which diffusion is so complete that it amounts to a "washing" of the membrane,* in this case the cornea. It seems likely that the radiant heat acts both as a catalyst for the chemical process of dialysis, and as a therapeutic agent acting upon inflammatory areas.

Summary

It is suggested that Glaucoma follows the presence of excessive albuminous material in the aqueous (a degenerative condition), this

*It may be that the further action of hydrolysis occurs at this time upon the fats deposited within the cornea, and heat and alkalines breaking up the fat (in the presence of water) into an acid and glycerine, e.g., C_3H_5 ($00C.C_{15}H_{31}$)$_3$+3 H_2O= $3C_{15}H_{31}$. $C00H$+C_3H_5 $(OH)_3$ thus facilitating the removal of the fat from the cornea by breaking it down, or synthesizing it into a more liquid form.
(fat) (water) (acid) (glycerine)

causing an undue or abnormal influx of lachrymal fluid through the cornea by diffusion, the end result being an increase of osmotic pressure within the eye, and at the same time filling the interstices of the cornea with albuminous and oedematous material, this in turn resulting in the cornea becoming cloudy or opaque from the presence of this translucent material; that secondarily, but *of only coincidental importance,* there is a gumming up, or partial blockage of the spaces of Fontana with thickened, colloidal aqueous humor.

It is further suggested that radiant heat used in conjunction with a solution of alkaline crystalloids and glycerine acts as a catalyst for equalizing the two-way process of diffusion, and as a therapeutic agent in the treatment of any inflammation present, this procedure resulting in the more or less immediate relief of intraocular pressure and pain, and the subsequent and total removal of albuminous and oedematous materials from within the parenchymata of the cornea by dialysis, and that this latter removal may be due in part to the hydrolysis of fatty deposits within these interstices.

Conclusion

The percentage of cures in Glaucoma following surgical interference methods is rather lamentable. This seems more natural an outcome when we consider that surgery in this disease is purely symptomatic treatment, and does nothing to clear up the condition responsible for the pressure increase in the beginning. Fuchs finds that many patients are never free from pain until the entire eye atrophies into a *pthisis bulbi,* one acute attack following another with increasing severity until the eye is lost in spite of all treatment measures. (4)

We feel that further experimentation along chemical investigative lines as to the true etiology of Glaucoma is indicated. While we feel personally that there can be but one answer to this problem, any final solution must be proved beyond all peradventure of a doubt in the chemical-pathologic laboratory, and suggest that this work be carried out by the staff in the Pennsylvania State College of Optometry.

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THE ACCEPTED STANDARDS OF CONVERGENCE REQUIREMENTS ARE INSUPPORTABLE*

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In view of the universally accepted belief that people of the present generation have not inherited an adequate ocular apparatus to meet the demands of modern civilization, it would seem reasonable to question any standards for visual requirements founded on a cross section of so-called normals, because they lack inherited qualification.

I believe the standard for convergence requirement adopted by the Graduate Clinic Foundation is 19 P. D., for 6 meters, and 21 P. D. for 40 centimeters, and that these figures were based upon an average of the convergence abilities of some thousands of subjects showing no evidence of asthenopia or uncorrected refraction and engaged in various pursuits and being of various ages.

Since we are all agreed on the belief that civilization's demands upon the function of vision have advanced far beyond the visual development, and its inherited qualities, such a choice of subjects could only develop what to expect, not a standard of requirement to meet the functional needs for modern life.

If the subjects had been picked from individuals engaged in close range visual pursuits, each of whom showed no evidence of asthenopia nor uncorrected refraction, it is my relief that the average would have been somewhat higher. However, I doubt if adequate standards could be derived from even such a group because of uncontrollable variables necessarily present. Particularly I would call attention to the fact which all investigators have observed, that an individual of high vitality often exhibits no symptoms of asthenopia even when his refraction is quite below normal standards, therefore with this certain knowledge an individual of high vitality and normal refraction undoubtedly could carry considerable functional disorder without complaint.

Upon these considerations it would seem that the present widely accepted standards for the function of convergence are based upon an untenable theory, or foundation.

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But we must have a standard for convergence requirement to meet present day needs, and that standard must be higher than heretofore necessary, so since we cannot depend upon an average of selected subjects we must turn to physiology, psychology and philosophy and develop a theory that will stand the test of clinical research.

Unquestionably the high demands upon the function of vision fall upon convergence and accommodation, a subject that has been widely discussed for many generations, and about which many conflicting opinions and theories have been advanced.

Accommodation is accomplished by the contraction of the cilliary muscles, which are of the smooth non-striated type; requiring nerve impulse to contract; which hold contraction for long periods of time; which give little evidence of fatigue; which require nerve impulse to relax.

Convergence is accomplished by contraction of the medial rectus muscles which are of the skeletal striated type; which require a chain of nerve impulses to maintain contraction; which fatigue relatively very rapidly; and which relax by a chemical process. Fatigue resulting in the fibers becoming engorged with lactic acid.

Howell states under the discussion of Distinctive Properties of Smooth or Plain Muscle, "The simple contractions of plain muscle have a very long latent period that may be a hundred or five hundred times as long as cross-striated muscle, and the phases of shortening and relaxation are similarly prolonged."

Horton states that "but one impulse per six seconds is required for full tetanic contraction of plain muscle, while from fifty to one hundred impulses per second are required for cross-striated muscle." These two statements are therefore inversely in virtual agreement, and many other texts which I have searched give approximately the same information. A helpful, though abridged discussion of muscle innervations, can be found in Horton's article in the 1935 New York Optometric Year Book.

For many years it has been found necessary to allow a 50% reserve for the function of accommodation. At the average age of 45 years for the onset of presbyopia the accommodation total is three and one-half diopters, and we must supply a plus one lens to provide comfortable reading at one-third meter. At that distance his requirement is three diopters of accommodation, leaving him but one-half diopter for reserve, which is not sufficient. Adding one diopter brings his reserve to one and a half diopters, which is 50% of the required three diopters. But according to the physiology and neurology of the muscles involved in this process accommodation is much less fatiguing than its concomitant partner convergence. Then it would seem logical that convergence must have much more than a reserve of 50% to continue to function with it for any extended period of time without fatigue.

Use of the eyes at ⅓ meter requires 18 P. D. of convergence for a subject with a pupiliary width of 60 millimeters. The Graduate Clinic Foundation standard for convergence is 21 P. D. (there is but a fractional difference at 40 centimeters), leaving for reserve but 3 P. D. or 16⅔% of the effort required. In the light of reason this cannot be an adequate reserve. A person with high vitality may be able to get away with it for a considerable time, perhaps some years, but the strain will be cumulative, the result being any one or more of a chain of nervous involvements probable, if his demand on convergence is in accord with modern times.

Dr. T. J. Arneson of Minneapolis reported a few years ago that in his clinical experience he had found that a convergence of 40 P. D. at 6 meters with a circular moving test object was a necessary standard. Later Dr. H. S. Horton, head of the eye department of East Cleveland Clinic and of the Optometric Research Group of Cleveland, established through clinical experience that the standard for convergence requirements should be 35 to 40 P. D. at 6 meters, the test object being a *stationary* row of vertical letters. The results of his investigations are reported in his work on Phoria and Duction Analysis, and his article in the 1935 New York Year Book.

Much credit may be given Dr. Arneson for his early discovery that a high standard of convergence at 6 meters must be available, but I cannot agree with his making 40 P. D. an imperical standard for all subjects, although it is approximately correct for the average person. It is my belief that a standard must be plotted for each subject in accordance with his pupil width, actual position of rest with regard to parallelism of the visual axes, and his habitual convergence requirements. Thus a 60 P. D. subject whose visual axes can be at rest when they are parallel will require a convergence of 18 P. D. for a distance of ⅓ meter, and with an equal amount for reserve will bring his standard to 36 P. D. and this amount of convergence must be available at 6 meters with a stationary test object where accommodation is not brought in to drive the convergence. At the near point he should be able to overcome a like amount of prism base out, and show a return to fusion of approximately his convergence requirement of 18. At the near point then he will actually converge 54 and return to fusion at about 36, because he is converging 18 P. D. before base out prisms are placed before his eyes. The refusion requirement may be somewhat flexible, but must be within 3 or 4 P. D. of 18.

Some may argue that accommodation is in force after a certain amount of prism base out has been placed before the eyes while testing the convergence at 6 meters. Accommodation is called in, and it is of some importance to record that point, if it can accurately be determined, but accommodation here is of no assistance to convergence but probably a deterrent. If it enters the procedure too early the diplopia point is usually lowered. In testing the convergence available at the near point

accommodation is demanded and convergence should be greater, at least about as much as 18 P. D. the near point demand. Let me here call attention to the fact that many instruments in use are inadequate for a full test of this function, because the apertures are too small, the rotary prisms too far from the eyes of the subject, and the pupillary adjustment cannot be narrowed enough to keep the lines of vision in the lenses. Many low findings are therefore due to the visual lines encountering the margins of the prisms, especially when making the test at one-third of a meter.

In estimating the standard for convergence for a given subject no consideration should be given for any phoria findings, except hyperphoria which must be corrected while making the lateral ductions. Esophoria and excessive exophoria will usually yield to proper orthoptic training. Before each training period a record of the phorias and ductions to the diplopia points should be recorded, and if the questionable phoria does not yield a correction of the convergence requirement may be advisable, especially if an excessive exophoria persists, thus indicating that it is caused by malattachment of the extrinsics.

In the December, 1934, issue of the American Journal of Optometry a very ably presented article by Dr. Wm. J. Tait appeared, the purpose of which was to show that base in exercise would not develop abduction findings of a normal subject. According to our standards for convergence reserve requirements three out of the four of Dr. Tait's subjects would have proven ineligable for his experiment because each of the first three show insufficiency of convergence reserve, and if those young men had no consciousness of fatigue after hours of study they must, we believe, have been the fortunate possessors of vitality high enough to absorb the strain, and some day, we fear, they will know it.

It is my hope that a restudy of the requirements for convergence reserves to meet present day needs may result from this monograph. The standard of an equal reserve to the convergence required, as set forth, has thus far stood the test of clinical research conducted by our Cleveland group, and has proven very helpful in our efforts to relieve complaints of asthenopia.

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UNUSUAL USES FOR BIFOCALS

Dr. Mollie Armstrong in an editorial in the January, 1925 number of the TEXAS OPTOMETRIST relates the interesting experience of, after having prescribed bifocal lenses for a patient, having him return and request that she make him a second set up-side down. Naturally she was somewhat puzzled and on inquiry it developed that after his office hours he played an instrument in a band and when he was obliged to march, his music was above his eye level and to enable himself to walk he wanted to see the ground at his feet. The bifocals were prescribed as he requested and proved satisfactory for this purpose.

The writer, also, was recently called upon to provide something of unusual character, in bifocal form. The patient, male, a normal presbyope of 53 was a stock keeper in a large wholesale hardware house and his work necessitated him viewing markings on boxes and packages on shelves above his eye level, he also having some desk work to do. After some experimental work a pair of bifocals were prescribed for him. The greater portion of these lenses were O.U. + 2.00 D. Sph. Attached to this in the upper quadrant were a pair of —0.75 D. Spherical segments, which gave him a five-foot range of vision. The patient of course removed this combination when not at work.

A somewhat similar experience was also had with a barber, a Mr. A. age 63, who wanted an inverted bifocal to use at his work. He however did not wish cement bifocals, but could not stand the cost of the one piece bifocal lenses. His problem was presented to Mr. H. Hutchens, an optician, who suggested taking a pair of +3.00 D. Spherical P.C.X. lenses and resurfacing a small portion of each, making this portion a +1.00 D. Sph. in strength and using this at the top, it being his distance correction. This was done with the patient's entire satisfaction despite the vertical displacement that must have been present while looking through the small prismatic distance portion.

A third unusual bifocal recently came to the writer's attention. This pair of lenses was composed of a large pair of light amber plano goggle lenses on the inner surface of which had been placed a pair of small circular opifex bifocal segments. These segments were near the center of the lens a little below the driver's line of vision. These segments were so focused that the wearer could easily view the apparatus on the dash board of his car. The segments were 11 mm. in diameter.

C. C. K.

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OPHTHALMOSCOPY UNDER NATURAL ILLUMINATION

In making a bedside refraction the optometrist is frequently put to no little trouble to sufficiently darken the room so as to make an ophthalmoscopic examination of the fundus possible. Yet with any of the methods of ophthalmoscopy now in use with portable ophthalmoscopes, it is necessary to materially reduce illumination in order to get a distinct view of the retinal area.

Because of this fact and because of the difficulty sometimes encountered in attempting to darken a room, Posner[1] decided to convert his ophthalmoscope itself into a miniature dark room. This he accomplished by attaching two aluminum eye cups, one to each side of the revolving lens system of his ophthalmoscope. The inside of each cup was painted dull black after holes had been made in their respective bottoms which were then fastened to the ophthalmoscope, in such a manner as to permit the examiner to look through the eyecups as well as the normal openings in the instrument.

In using an instrument so arranged, the ophthalmoscope is merely placed over the eye to be examined with the edges of the eyecup firmly touching the skin covering of the orbit. The examiner next moves forward until he himself is in a similar position with reference to the other eyecup on the outer-side from the patient. Posner finds the distance about right and says that with a little practice one can easily adjust oneself

[1] Posner, C. A miniature Dark Room Device for Ophthalmoscopes. Jour. A.M.A. Vol. 105, No. 8, p. 586. 1935.

to the new arrangement. The idea sounds practical and should prove to be of value to optometrists who wish to improve their examinations made in the patient's home. C. C. K.

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REFRACTION ROUTINES

While optometrists will never agree on a proper routine in the matter of an ocular refraction, the writer believes the following outline of procedure, is a fair example of a minimum routine which should be carried out in each case.

Case History, Symptoms and Personal Hygiene
Visual Acuity Uncorrected
External Ocular Inspection
Ophthalmoscopic Examination
Perimetry and Stereo-Campimetry
Ophthalmometric Findinds
Skiametry Static at one meter
Skiametry Dynamic at one meter
Subjective Findings
Determination of the near point at which patient works
Skiametry Dynamic at this distance
Subjective Positive Accommodative Test
Determination of patient's near point
Subjective Presbyopic Addition if needed
Determination of the Dominant eye
Tonicity Test at six meters
Duction Test at six meters
Tonicity Test at thirty-three c/m
Reserve Fusion Convergence at thirty-three c/m
Color preception tests.

In all cases the above procedure should be followed with a post-refraction at which time the unusual features of the case can be further studied before the diagnosis is made and the prescription or treatment is decided upon. In some cases no prescription should be written until the case has been kept under observation for periods varying from ten days to three months, the patient using temporary treatment corrections during this period. C. C. K.

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RESEARCH, SO-CALLED

For several decades at a rather large number of universities some of the professors and graduate students in the department of Psychology have devoted no small amount of time and money to the carrying on

of various forms of ocular and semi-ocular research. Much of this research work has been seriously done and some fine results of marked value to us as optometrists and also to the public at large have resulted. Unfortunately, many of these workers have devoted themselves to problems of such doubtful value that regardless of the outcome of the research nothing of value could result and others have carried on their so-called work in such a spirit of merely doing something so as to get required credit, that the results of these researches have proved to be of little value whatsoever. In addition to this, much of the questionable result of these researches is dressed up in a lot of pseudo-scientific verbage and passed on to the public as scientific facts. A recent classic example of the type of piffle that can come from this source is that of a professor who is reported as being a psychologist of repute and one who is a noted student of the science of mental phenomena.

This psychologist is responsible for the following more-or-less interesting, but decidedly questionable data regarding the personal behavior charastics of people whose irises are pigmented as follows:

(1) Persons with dark blue eyes are apt to be very reliable.

(2) Persons with light blue eyes are frequently found with calmness of mind and an equanimity of temper but with less energy than those persons having dark blue eys.

(3) Persons with very pale blue eyes have very little feeling or emotion.

(4) Persons with gray eyes are very imaginative.

(5) Persons with steel gray eyes are usually hard, cold and lacking in sympathy.

(6) Persons with brown eyes are usually affectionate and very unselfish.

(7) Persons with brown eyes whose irises are flecked with green are very courageous.

(8) Persons with hazel colored eyes are apt to be inconsistent.

(9) Persons with green eyes are frequently found to have quick tempers and strong imaginations.

From the foregoing we strongly suspect that this learned professor has been reading too many novels and we would also be safe in assuming that he has dark blue eyes himself while his mother-in-law is sure to be green-eyed. The depression from which we are passing leaves little to be thankful for, yet should it curtail the output of psychological nonsense similar to the above it would leave us something indeed to be grateful for. When university professors have time to waste in speculation of this character it is high time for the heads of academic bodies to curtail their teaching staffs to the bone. C. C. K.

CORRESPONDENCE

ARGYLL ROBERTSON PUPIL*

Thomas G. Atkinson, M D.
Chicago, Ill.

I read with interest an article in your July issue by Dr. Frederick W. Brock, on the Argyll Robertson pupil.

As a general discussion of irresponsive pupils, of which there are several types with several causes, it was very instructive. But all irresponsive pupils, even those that respond to accommodation but not to light, are not Argyll Robertson pupils. According to modern symptomatology, a genuine Argyll Robertson implies a very definite syndrome, with an equally definite lesion topography. The syndrome is as follows:

Retinal sensitivity to light.
Contracted pupils.
Immobile pupils, except to accommodation.
Undilatable by atropine or by painful skin stimuli.

Such a syndrome is possible only with a lesion which involves two separate neural pathways, viz., the light reflex pathway from the retina to the third nerve nucleus to the iris, and the sympathetic pupillodilator fibers, from midbrain to cervical sympathetics to iris.

The lesion can have only one topography, namely, in the midbrain, around the aqueduct of Sylvius, where, in their course from the hypothalamus to the spinal cord, the sympathetics run for a short distance very close to the pupillary light reflex fibers. It is usually, but not invariably, a luetic lesion. No other type of irresponsive pupil is a genuine Argyll Robertson pupil.

DR. THOMAS G. ATKINSON,
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REFRACTIVE QUERIES

CALCULATION OF LOSS OF VISION

To the Editor: I have been puzzled for some time to know the method whereby industrial loss of vision is computed. Please give me references

*Submitted August 9, 1935.

which will enable me to study up on this matter.—W.S.C., Chicago, Ill.

ANSWER—Dr. A. C. Snell found as the result of clinical investigation that total visual incapacity lay somewhere between 20/200 and 2/200, binocular visual acuity. (Jour. A.M.A. Oct. 31, 1925, p. 1367). There is, however, considerable difference between visual inefficiency and ocular industrial blindness, the latter being based on (1) visual acuity, (2) the size and shape of the visual field, and (3) the ability of the eyes to satisfactorily turn in their orbits. The full table with a consideration of the various factors involved in the determination of industrial visual efficiency was presented by J. E. Lebensohn in Archives of Ophthalmology, July, 1933. For details of the calculations the article entitled "Percentage Evaluation of Macular Vision" by A. C. Snell and Scott Sterling which appeared in the September issue, 1925, of Archives of Ophthalmology will give all the details. It is interesting to note that most states have ruled that a visual acuity of 20/200 is industrial blindness although mathematically this visual acuity represents a general visual efficiency of 20%. In this connection we understand that Test Charts can be procured from the Bausch and Lomb Company of Rochester, N. Y., that, along side of the distance notations, have the A.M.A. ratings of visual efficiency. C. C. K.

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BOOK NOTICES

MODERN OPHTHALMIC LENSES AND OPTICAL GLASS.—Theo. E. Obrig. Published by the Author. 7 East 48th Street, New York City. 323 pages, 168 illustrations. Cloth binding, 1935.

Obrig in his book "Modern Opthalmic Lenses and Optical Glass" compiled a considerable amount of material of value to all those interested in Optics and particularly those interested in refraction. At first he gives an historical account of glass making. He describes in detail the different processes used in the manufacture of glass, the chemical and physical characteristics of different types of optical glass.

One chapter is devoted to colored lenses, the effects of radiant energy on the eye and suggests the prescribing of certain types of absorbtion lenses in special conditions of functional and organic disturbances. He describes clearly in one chapter "contact lens" and the technique of their application, the telescopic spectacles both the Zeiss and the Feinbloom types. The technique of testing subnormal vision with Zeiss and Feinbloom types of telescopic lenses is briefly described. This is to be regretted for there is a need for a more elaborate explanation on the technique of testing, prescribing and fitting of these special devices to the subnormal vision patients.

The author discusses at some length Iseikonic lenses and describes the Ophthalmoeikno-meter (an instrument to measure the differences in size of image) the magnification of the iseikomic lenses is described, refrences are also given to places where the original material was published, so that the reader, who may be interested to study this subject more thoroughly, may be able to go to the original sources.

The last part of the book is devoted to a discussion of the different types of bifocal and trifocal lenses with suggestions as to what kind of a lens is best to prescribe for special types of cases. Obrig's book is a valuable contribution to ophthalmic optics. J. I. K.

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